MAR 14 1921

Minneapolis Clinic Week, April 25-30, 1921.

VOLUME IV NUMBER 3

MINNESOTA MEDICINE

Journal of the Minnesota State Medical Association

EDITOR

CARL B. DRAKE, M. D., St. Paul

ASSISTANT EDITORS

STANLEY R. MAXEINER, M. D., Minneapolis PAUL D. BERRISFORD, M. D., St. Paul

ASSOCIATE EDITORS

First District
GEO. S. WATTAM, M. D., Warren

Second District
A. W. IDE, M. D., Brainerd

Third District
E. L. TUOHY, M. D., Duluth

Fourth District
F. L. ADAIR, M. D., Minneapolis

Fifth District
GEO. B. WEISER, M. D. New Ulm

Sixth District
A. E. SPALDING, M. D., Luverne

Seventh District
H. B. AITKENS, M. D., LeSueur Center

Eighth District
A. F. SCHMITT, M. D., Mankato

MARCH, 1921

CONTENTS

ORIGINAL ARTICLES

JOSEPH L. MILLER, M. D.—Some of the Problems of Internal Medicine..... 119
FREDERICK L. HOFFMAN, LL. D.—Some New Aspects of the Problems of Cancer Control 124
H. Z. GIFFIN, M. D.—The Present Status of Splenectomy as a Therapeutic Measure 132
WILLIAM J. MAYO, M. D.—Hemolytic Icterus..... 138
C. EUGENE RIGGS, M. D.—Three Neuropsychiatric Notes Epilepsy, Lethargic Encephalitis and the DeMentia Precox Syndrome..... 141
T. L. CHAPMAN, M. D.—Factitious Toxicity of Previously Simple Goitres..... 148
ROSCOE C. WEBB, A. B., M. D.—Fractures of the Base of the Radius..... 151
W. E. SISTRUNK, M. D.—Mixed Tumors of the Parotid Gland..... 155
ERNEST T. F. RICHARDS, M. D. and EDGAR T. HERMANN, M. D.—Polycythemia Vera 161
L. W. MORSMAN, M. D.—Uses and Advantages of Electrically Driven Bone Surgery Equipment in Head Surgery, with Particular Reference to Ear, Nose and Throat Operations 166
GEORGE DOUGLAS HEAD, M. D.—Acute Lymphatic Leukemia..... 169

(Continued on Advertising Page III.)

Owned and Published Monthly by

THE MINNESOTA STATE MEDICAL ASSOCIATION

BUSINESS MANAGER

J. R. BRUCE, 403 Central Bank Bldg., Saint Paul

Telephone: N.W. Cedar 1688

Entered at the Post Office in Saint Paul as second class mail matter.
Accepted for mailing at the special rate of postage provided for in section 1103, act of October 3, 1917, authorized July 18, 1918.

Subscription Price { $3.00 yearly, Domestic
$3.50 yearly, Foreign

Riggs Peerless Neutralizer

Something every Ophthalmologist should have

(Picture Actual Size)

The Only Satisfactory Device for Neutralizing the Lens and Determining the Axis

A new development in lens testing equipment. The biggest thing brought out since the perfection of the lens measure. A simple, inexpensive device that makes possible quick and accurate results in neutralization of lenses and testing of axis.

Riggs Peerless Neutralizer is made to hold lenses of any size or shape—oval, round or drop eye, mounted or unmounted—and any style of frame or mounting, spectacle or eyeglass, whether of metal or of shell.

The revolving cell with spring clips for three trial rings enables the operator to make up any combination desired, from the test case; and the axis scale indicates the axis exactly, eliminating all guess work.

To operate, simply place lens in holder; put proper test lenses in clips; revolve cell until neutralization is obtained and read from axis scale the position at which axis is cut.

Riggs Peerless Neutralizer saves time—insures accuracy—removes all doubt. It should be in every trial case. The price is only $9.50 net. No one can afford to be without it.

SHALL WE SEND IT BY RETURN MAIL?

RIGGS OPTICAL COMPANY

Equipment Specialists

FARGO, **SIOUX FALLS,** **HELENA,** **SIOUX CITY,** **SPOKANE,**
OMAHA, CEDAR RAPIDS, LINCOLN, QUINCY, PORTLAND, SEATTLE, TACOMA, HASTINGS

CONTENTS—Continued from Title Page

EDITORIALS:

e in Mal-practice Suits..... 183
e Late Osteopathic Bill..... 183
ite Solidarity..... 185
e Taking..... 184

S OF THE HOSPITALS..... 185

OBITUARY 186

REPORTS AND ANNOUNCEMENTS OF SOCIETIES 186

OF GENERAL INTEREST..... 186

NEW AND NON-OFFICIAL REMEDIES..... 188

PROGRESS 189

BE CAREFUL

When You Use

BEEBE BIOLOGIC PRODUCTS

It is recommended, to those who have never used BEEBE BIOLOGICS, that the directions be followed explicitly. Bear in mind that BEEBE BIOLOGICS are not like the ordinary biologic products.

BEEBE BIOLOGIC PRODUCTS are prepared from freshly isolated cultures of HIGH ANTIGENIC VALUE and therefore are much more active than the average and must be used according to directions.

IN THE TREATMENT OF PNEUMONIA

the physician SHOULD BE PREPARED TO ADMINISTER

Beebe Pneumococcus Antigen No. 15

on the first visit

Many surgeons are finding that BEEBE PNEUMOCOCCUS ANTIGEN No. 15 is of great value when administered at the time of operation as a protection against **ether pneumonia.**

BEEBE LABORATORIES, INC.

ST. PAUL, U. S. A.

For Future Reference

Physicians' Exchange New Telephone Numbers

From:

St. Paul - Nestor-1852
Minneapolis 0-Nestor-1852
or Dinsmore-1677

(No charge for calling these numbers from either city)

May we explain how the service of the Physicians' Exchange is valuable and economical to physicians and patients? The cost is insignificant.

ARE YOU A MEMBER?

YOUR
CONFIDENCE

in our results forms the foundation of our success. Such confidence is engendered only by reliable results and prompt reports. These are made possible by our ***specialization*** in clinical laboratory diagnosis. May we not serve you?

Culture media and containers sent upon request.

THE DEEDS LABORATORIES

BESSE BUILDING
SIXTH AT NICOLLET
MINNEAPOLIS

MINNESOTA MEDICINE

Journal of the Minnesota State Medical Association

VOL. IV MARCH, 1921 No. 3

ORIGINAL ARTICLES

SOME OF THE PROBLEMS OF INTERNAL MEDICINE*

JOSEPH L. MILLER, M. D.
Chicago, Ill.

The only member of our profession who is satisfied with his knowledge of medicine, and who considers it a well rounded out and thoroughly understood science, is the medical graduate at the time he receives his diploma. He has studied diligently the text books placed before him and memorized the notes taken at his lectures. He has creditably passed his examinations and thus fulfilled the requirements demanded by his Alma Mater.

The graduate today has, however, a much humbler opinion of himself than did we who graduated a quarter of a century or more ago. This is due in part to his more extensive premedical training and in part to the changed character of his teachers. For a teacher today to admit to his students that it is impossible to explain many of the phenomena of disease, is not considered as evidence of ignorance; furthermore, a larger percentage of our teachers have taken an interest in the investigative side of medicine and thus aroused the interest of the student in its unsolved problems.

Valuable additions to our knowledge of medicine have not been contributed necessarily by the physician with extensive hospital or laboratory facilities, but rather by the alert and independent mind unwilling to abide by tradition.

The lapse of time rather than diminishing, has increased the problems in medicine. The blazing of trails has opened up new vistas with innumerable ramifications. There has never been a time in history when the problems in medicine were so numerous and diversified and so appealing to the varied or special qualifications of all those with an inquisitive turn of mind or an interest in public service.

If we review hastily the various diseases with which we come in contact, in how many of these is out knowledge of etiology, prevention and curative treatment, reasonably complete? There is only one disease, malaria, where we can express a high degree of satisfaction with the completeness of our information. Its etiology has been definitely determined, marked progress has been made in practical preventive measures and the curative treatment well established. Yet in this disease, Bass within the past two years has shown that the customary methods of administering quinine fails in the majority of cases to elminate the parasite. Pursuing his studies further he has determined the minimum daily dose of quinine and the length of time it must be continued in order to completely eliminate the infection.

Before discussing further the limitations of our knowledge, it might place us in a better frame of mind if we review briefly some of the large problems in medicine which have been attacked in recent years with most gratifying results.

The combating of typhoid by sanitation and protective inoculation with the saving annually of thousands of lives and millions in money, is recent history more or less familiar to all. It has been shown that epidemic typhoid is preventable. The city or community which suffers from repeated epidemics of typhoid can justly be accused of gross negligence. It is gratifying to note that there has been a steady and marked reduction in the incidence of typhoid in the cities of the United States until at present we have a record which compares favorably with that of Europe. The most striking illustration of what has been accomplished may be gathered from the prevalence of typhoid in the United

*Read before the Minnesota State Medical Association, St. Paul, October, 1920.

States Army during the Spanish American and the recent World War. The total number of cases of paratyphoid and typhoid in the entire American Army at home and over seas from August, 1917, to June 1, 1919, was only 1,418 with 158 deaths. Compare this with 2,683 cases and 248 deaths occurring among less than 12,000 troops at Jacksonville, Florida, during the Spanish American War. This has been accomplished in only two decades. A short period of time to have conquered a scourge which in previous wars has been more deadly than bullets.

Yellow fever, within the memory of most of those present, was a pest in the Southern states costing thousands of lives and inestimable economic loss. The epidemic of 1878 took a toll of 13,000 lives in the Mississippi Valley. It was a still greater plague in the West Indies. The French on the Island of Haiti were almost exterminated in 1842. During one epidemic half the population of Guyaquil in Ecuador was sacrificed. It was largely responsible for De-Lesseps' failure to complete the Panama Canal. For years its presence in South America had been a menace to commerce. When the United States acquired Cuba, they received gratis a hot bed of yellow fever. Proper development of the Island was impossible until this disease was brought under control. A group of army officers were selected with Dr. Walter Reed in charge to study the manner of dissemination and means of exterminating the disease. Their method of attacking this problem should serve as a model for constructive scientific investigation. They definitely determined that a certain species of mosquito was the sole carrier of this disease. By destroying the breeding grounds of the mosquito and carefully protecting the sick by screening, they were soon able to reduce greatly its prevalence and by following up these methods, yellow fever has finally been eliminated from Cuba. The application of the knowledge gained by this small group of medical men, made possible the construction of the Panama Canal. In recent years the International Health Board of the Rockefeller Foundation has extended this war against yellow fever and in many of its strongholds in South America it has been completely exterminated. Noguchi, working with this Foundation, has recently discovered the specific microorganism and prepared a serum with both protective and curative properties. The recent death of General Gorgas prevented him from "writing the last chapter of yellow fever."

A very important campaign has been carried on for several years by this same Board in combating malaria. In the North we are prone to consider malaria as a rather unimportant disease. Throughout the world, however, 800,000,000 people live in tropical and subtropical regions where malaria is more or less prevalent. In India alone it is computed that 1,300,000 die annually from malaria. In our Southern states not only is malaria responsible for many deaths, but it lowers the efficiency of a large group of individuals and has prevented the development of large areas of tillable land. The Rockefeller Foundation selected for their educational campaign several towns in Arkansas. They were able to show that the incidence of malaria could be reduced almost 70 per cent at a per capita cost of sixty cents. This was accomplished by destroying the breeding places of the mosquito, by proper screening and through the use of immunizing doses of quinine.

Bubonic plague during the last quarter of a century has been brought to our shores repeatedly, especially on the Pacific and Gulf Coasts. It is highly probable if this had occurred fifty years ago, it would have developed into a widespread epidemic. Thanks to our knowledge that the rat and gopher harbor the germ, it has never been able to obtain a foothold.

We are all familiar with the hookworm campaign in the Southern states and some of the islands of the West Indies. Ninety per cent of the inhabitants of Porto Rico were infected with this parasite. By means of educational campaigns this disease is gradually being brought under control.

Intensive studies have been made in many other diseases, but up to the present have borne little fruit. In this last, a notable example is cancer. This labor and money has not, however, been lost as it has paved the way for future investigators who, let us hope, may finally reach the goal.

Turning to the problems still unsolved, their name is legion, and to attempt to discuss them in detail would require a review of all of medicine. When we consider the long list of con-

tagious diseases we are depressed by the limitation of our knowledge. In several the etiological agent has not been discovered. Means of dissemination are still far from determined and preventive measures are still in an unsatisfactory state. We cannot boast of the value of therapeutic measures as evidenced by most of these diseases being still referred to as self limited in duration. The recent epidemic of influenza was the most serious that has ever been recorded. We must frankly admit we were helpless in preventing its spread or in treating the disease after it had developed. Even in army camps with their rigid discipline and ability to enforce every precaution, the disease entered undaunted and took its toll.

When we consider the long list of chronic diseases as chronic arthritis, nephritis, myocarditis, arteriosclerosis, hypertension, pseudoleukemia, leukemia, pernicious anemia and diabetes, we are humbled by the limitations of our knowledge. Their etiology is still hidden in mystery. Preventive measures and curative treatment are renowed only by their absence. We are valiantly struggling with these diseases and from time to time valuable acquisitions to our knowledge are recorded.

It should be strongly emphasized that the acquisition of new knowledge is not restricted to our research institutions or to the physician who has access to large clinical hospitals. MacKenzie carried on a large part of his investigations on the heart, which have made his name famous, on private patients and with little aid from hospital facilities. Corrigan in his masterly studies of aortic regurgitation had available only six hospital beds. Recall the work of Beaumont, whom Osler fittingly refers to as the "backwoods physiologist," in his observations on the stomach of Alexis St. Martin. His untiring efforts over a long period of years, without facilities and with a refractory patient should encourage each of us to do his bit. To be an investigator, it is only necessary to seek the truth. to see things through our own eyes and to record these observations accurately. Such observations must survive because they are true. Inaccuracies of observation, or distortion of vision in the effort to prove a theory, is responsible for many of the fallacies and passing fads in medicine.

It might be of interest to take stock of our actual knowledge of some of the more common diseases with the view of reminding us of its limitation.

Pneumonia, a disease responsible for 70 per cent of the deaths from disease during the recent war, is one of our medical problems. The microbic agent is usually the pneumococcus or streptococcus and possibly occasionally the influenza bacillus. Our knowledge of the predisposing agencies is still exceedingly limited. Close contact when the disease is prevalent increases the liability of infection, but all exposed contacts do not contract the disease. Exposure to cold, formerly thought to be such an important factor, has lost much of its significance, as shown by severe epidemics of streptococcus pneumonia during warm weather. At least in large cities, epidemics of pneumonia of moderate extent during the summer months are not unusual. A considerable number of other predisposing factors might be mentioned, to none of which, however, can we attach great importance. In fact, comparatively little progress has been made during the last half century in the prevention of this scourge. We were stimulated for a time to hope that protective inoculation might solve this problem, but the most recent investigations indicate the present day methods of protection are so transitory in character as to render them almost valueless. Happily, however, the future is still before us and we should not give up hope that eventually aid from this source may be secured.

In the treatment of pneumonia as shown by statistics, comparatively little advancement has been made. In drug therapy nothing of significant value has been discovered. Serum therapy is still in its infancy, but up to the present time we are forced to admit the best of the sera are of doubtful value. This rather dark picture should not lead us to lose hope that another Pasteur or perhaps some obscure physician student may discover a remedy for this dread disease.

Typhoid fever has shown a most gratifying decrease during the last quarter of a century due to the discovery of practical preventative measures. When we consider, however, the treatment of the disease we have little advantage over our forefathers. The value of the Brand method of hydrotherapy has probably

been greatly over estimated, as is shown by refering to quite extensive control statistics soon after this method was introduced. The lowered mortality in recent years is probably due at least in large part to improved diagnostic measures which enable us to recognize mild cases of this disease. The introduction of the more liberal diet has increased the comfort of the patient and probably reduced slightly the mortality. We must still admit, however, a mortality of 10 per cent and to further reduce this is one of the present day medical problems.

The chronic arthritides present practically the same difficulties from a preventative and especially from a therapeutic standpoint as they did when this disease was first described. The early hopes that vaccine therapy and removal of focal infection might offer hopes of cure or at least stop the progress of the trouble, has unfortunately not been sustained although probably of some value. He who discovers a remedy for this disease will earn the everlasting gratitude of a host of sufferers.

Chronic hypertension is another of the live problems in medicine. With the exception of a small group of cases, we are quite at a loss to explain the origin of this condition. We are still much confused as to cause and effect. It is, therefore, needless to say that preventative measures are in the purely theoretical stage. There is little or no evidence to show that diet, worry or overwork is responsible for chronic high blood pressure. Methods of treatment are equally unsatisfactory. It is probably true that certain measures may prevent or delay the complications of hypertension, but there is no method of treatment now in use that will permanently lower chronic high blood pressure.

Pernicious anemia, leukemia and pseudoleukemia form a group where our knowledge outside of pathology and symptomatology is most meagre. We know little of the etiology and nothing of prevention in these diseases. Treatment is at most merely palliative. It increases the patient's comfort and prolongs life, but does not effect a cure. Each of these diseases annually takes a considerable toll of lives and offers an excellent field for the investigator.

We might pass thus in review, many other diseases in which our knowledge is of the most primitive character. New facilities for study, the greater ease with which money can be secured for investigation in medicine and the increasing number of well trained workers in the field, promise well for the clearing up of many of these obscure problems.

Diverging somewhat from what is ordinarily considered the science of medicine, there are other problems no less important when we consider medicine as the art of healing.

Social service, a relatively new and unappreciated branch of medicine, deserves consideration. The physician, especially in public medical service, has considered his duty completed when he has restored the individual to a sufficient degree of health to enable him to return to his daily vocation. It may have required weeks of rest in bed, nursing and medical care to restore the compensation in a patient with heart disease. All medical supervision or interest in the patient is gone when he passes through the door of the institution which has given him such excellent attention. He returns to the same employment in which he previously acquired his disability. Almost certainly within a few weeks he will again be unfitted for work, return to the hospital where he is kept for weeks until improved. This procedure is repeated until eventually even improvement in his condition is no longer possible and death relieves him of his misery and perhaps leaves his family a public charge. The duty of the social service or public welfare organization, is to secure for such a patient a position suited to his disability. He and his family are visited and instructed in the proper after care, and so secure complete cooperation at home. The majority of these patients will welcome interest of this character and when once established will be fully appreciative. This, as can readily be seen, is not merely a medical, but an economic problem. Patients with ailments where rigid dieting is necessary, can be greatly helped by instruction given to the wife or mother. The young mother needs instruction and kindly help in rearing her child. aid which must be extended much beyond the period of stay in the hospital. Dr. Cabot relates a striking instance of the limitations of medical treatment when confined to the walls of the hospital or dispensary. It was discovered on consulting the dispensary records that various members of a family had been coming to

the dispensary for more than a year to be treated for scabies. As several members of the family were infected, no sooner was one cured than he became reinfected and thus this endless chain was maintained. A few visits of a trained worker to the home accomplished in a brief period of time what the dispensary had not been able to do in a year. This may be an exaggerated example, but nevertheless much of our work is performed in this same thoughtless and inefficient manner. It is true the physician cannot be expected to perform this work himself, but he must be the moving spirit to stimulate the public to provide ways and means for carrying it on. A few hospitals have a well organized social service. Acquaintance with the patient begins in the hospital and when he is ready to leave plans are already completed as to his future care. Every community has a number of organizations of a charitable character. The duty of the social service worker is to discover the patient's needs and bring him in contact with the proper existing agencies which thus assume responsibility for his future care, whether this be securing suitable employment, a further rest in a convalescent home or the supplying of food, clothing or money to tide over an emergency. The social service department, however, still maintains an interested supervision by a follow up system. Many hospitals and dispensaries maintain an educational return clinic where at stated intervals in the evening or on Sunday, groups of former patients suffering from similar diseases may return to be looked over and talked to in regard to the precautions they should observe. These measures lead to a degree of permanency of cure not to be accomplished by other means.

Closely related to this work is vocational education or retraining of the handicapped. The importance of such training was abundantly emphasized with the disabled soldiers. Thousands of cripples from injury or disease develop every year in civil life. Such individuals, if poor, become wards of the municipality or the state. Others secure positions where they eke out a mere livelihood. A large percentage of these people, by careful selective vocational training, could at comparatively little cost not only become self supporting, but in many instances develop an earning power greater than before their disability.

This is a duty which belongs to the community, municipality or state, but the physician is the individual who can and should arouse public interest in this long neglected problem. Eventually the state will probably become responsible for this work. Meanwhile the community by example can hasten the day.

In conclusion, one other subject deserves consideration and it is broached with some hesitancy. There has been, especially during the past few years, much discussion on the question of health insurance. The man of family witn moderate income must figure very closely upon all his fixed expenditures as rent, clothing, and food. Perhaps his plans include laying a certain sum aside for a rainy day. Sickness is an unknown factor. He cannot determine in his annual budget the amount required for physicians' bills and perhaps hospital expense. For this reason illness finds him financially unprepared or if he has been thrifty, may easily consume several years' savings. Is it asking too much that the state should be interested in ways and means by which for a fixed sum, he or his family if ill, may be assured of satisfactory medical attention? Unfortunately in all plans now in operation, with the possible exception of certain large corporations, the sum allowed for this purpose is entirely inadequate to give the individual high class medical care. The profession as a whole, both in this country and in Europe, are opposed to health insurance in its present form as it does not give the individual the character of service which he deserves. Those members of the profession, and they make up the great majority, who are interested in the health and welfare of the people with moderate means will not oppose health insurance when they are assured that it means better attention for the sick. First class medical service is time consuming and, therefore, expensive and cannot be secured without proper compensation to the physician. Good medical care very frequently requires the cooperation of several specialists, each of whom must be adequately paid. Unless adequate satisfactory fees are provided for, this work will be performed by those least competent to earn a livelihood in open competition.

It would appear that in order to carry out

such a plan most economically, centralization is necessary. There is a plan now being considered in England which is based upon such a foundation. District zones are established each with its health center which contains laboratories, consulting and operating rooms and a small number of hospital beds. This center is the headquarters of the medical staff which includes the various specialists. In other words, it should be a well equipped and well manned out-patient department with the addition of a limited number of hospital beds. In such a scheme, much of the work now done by the physician could be performed just as efficiently by less highly trained and consequently less costly labor. Properly equipped, such a center would attract especially able young men who would thus secure an opportunity to practice medicine under satisfactory conditions. It would thus not only serve the ailing public, but would also be an educational institution for the medical profession. The innumerable details of such a plan will eventually be worked out. Difficulties and opposition no doubt will arise. Eventually, however, it is probable some plan of this character can be adopted as it is practical and not a Utopian dream. Money in large amounts will be required to properly finance any satisfactory form of health insurance. A nation which spends millions of dollars annually for the prevention and treatment of animal and plant disease, should be educated to see that human life is even more valuable and it cannot be safeguarded without liberal expenditures of brains and money.

In this brief review only a few of the problems in medicine have been mentioned. It suffices, however, to show that medicine is a living science still in its infancy and offering splendid inducements not only to the scientist, but to those interested in the large problems of public welfare.

SOME NEW ASPECTS OF THE PROBLEM OF CANCER CONTROL*

FREDERICK L. HOFFMAN, LL. D.
Third Vice President and Statistician, The Prudential Insurance Company of America.
Newark, N. J.

Mr. Chairman, Ladies and Gentlemen:

I have been requested to take the place of Dr. Gaylord of Buffalo, who is unavoidably prevented from being present. I share your regret that we could not have heard Dr. Gaylord, who is one of the best informed experts on the cancer problem today. Dr. Gaylord has measurably advanced the cause of cancer research, as Director of the New York State Institute for Malignant Diseases, and he has brought to bear upon his work a strictly scientific attitude, indispensable in all inquiries aiming primarily at the truth and not made to substantiate some preconceived theory or point of view.

The cancer problem is as baffling today, if not more so, than it has ever been. The subject is becoming more ramified as research in one direction or another proceeds and as new problems arise, or new discoveries are made. No one can today lay claim to a thorough grasp of the whole subject further than in broad outlines, and even that is a task of considerable difficulty.

When the *Society for the Control of Cancer* was formed, it was clearly realized that the main objective of its propaganda should be to arouse the general public to the menace of cancer increase and the hopeful effects of early diagnosis and surgical treatment in the early stages of the disease. It was for this reason decided not to call the society one for the prevention, but rather for the control of cancer. In other words, the aim of the society is to bring about a reduction in the death rate from cancer as the results of early methods of treatment, whether surgical or otherwise. Nothing of value is known at the present time that would justify the belief that cancerous processes can be prevented: but the knowledge of pre-cancerous conditions leads to earlier diagnosis and treatment at a stage when the prognosis is generally quite favorable.

*Read before the Minnesota State Medical Association, St. Paul, Sept. 30, 1920.

Regardless of what the society has done, and it has done much, there has not as yet been a marked effect on the cancer death rate. In some localities where the campaign has been most effective, the rate has unquestionably declined, on account of cases coming earlier to operation. But, in a general way, it may be said that the vast majority of cancer operations still take place when the disease has reached an inoperable condition. Cancer is not only one of the most important causes of death, but *the disease is increasing* from year to year in practically all civilized countries. In the continental United States it may be conservatively estimated that during the present year there will not be less than about ninety thousand deaths from malignant disease. On the basis of an earlier estimate, the approximate number of deaths from cancer is divided somewhat as follows: Stomach and liver, 32,000; female generative organs, 12,500; peritoneum, intestine and rectum, 11,000 female breast, 8,500; cancer of skin, 3,500; cancer of the buccal cavity, 3,500; and of other organs or parts 13,000; a total of 84,000. This statement illustrates the wide diversity of the cancer problem and emphasizes the importance of considering the cancer question in detail, rather than in general terms, for methods of treatment applicable to one form of cancer may be wholly inapplicable, or partly so, to another form. The statement also illustrates the great importance of accurate death certification. It is lamentable that so many certificates should be sent in containing merely the term "cancer." It obviously is of the first importance that the organ or part of the body affected should be stated, for it will not advance the cause of cancer control to deal with statistics limited to cancer mortality in general terms. It, no doubt, entails a certain amount of additional labor on the part of the doctor to fill out a certificate in detail, but by doing so much subsequent and more burdensome correspondence is avoided. The filling-out of death certificates on the part of the attending physician is a statutory duty, which requires to be discharged in full cognizance of all that is implied in the subsequent use of the certificates for legal, medical or public health purposes.

The cancer death rate in the continental United States has been increasing from year to year, or at the rate of about 2.5 per cent per annum. There is no corresponding pesistent increase in the death rate of any other disease or group of diseases, and considering the vast amount of useful knowledge concerning the cancer problem, the increase reflects the lamentable apathy on the part of both the public and the medical profession towards the problem of the earliest qualified diagnosis, followed by the earliest radical method of treatment. Since the propaganda for cancer control is largely limited to cities or urban communities, it will be of interest to point out on the present occasion that the cancer death rate of twenty representative American cities increased from a rate of 72 per 100,000 in 1904 to 89 per 100,000 in 1914 and to 94 per 100,000 in 1918, not all the figures for 1919 being as yet available. *This increase is real and not apparent.* In fact, the true increase in cancer liability is much greater, for operative interference has in the meantime increased materially in efficiency. If it were possible to ascertain the number of successful cancer operations during the course of a year and by means of a follow-up system, the number of successful cures within a given period of time, it would become clearly apparent that the liability to cancerous affections is possibly today 50 per cent greater than it was fifteen years ago.

The control of the cancer death rate is *primarily a surgical question,* for unless the offending mass of cancerous tissue is promptly removed in the early stage of the disease, as all of you know, death is a foregone conclusion. It would be as sensible to argue against the installation of fire-preventive apparatus as to argue against a propaganda which will increase early surgical operation. There is the utmost urgency that the number of early operations should be very considerably increased as a first step in the more satisfactory solution of the problem of cancer control.

In *Minnesota* the cancer question is of special importance, particularly in the case of the Twin Cities. The rates, in fact, are so startling that they suggest the urgency of a special inquiry into determining factors which a qualified investigation might disclose. In the case of the city of St. Paul the cancer death rate has increased from 61 during 1904, to 74 during 1914

and to 113 per 100,000 of population in 1919. The cancer death rate of Minneapolis has increased in about the same proportion, or from 58 in 1904, to 93 in 1914 and to 106 per 100,000 in 1919. The St. Paul and Minneapolis rates are considerably above the average for the country as a whole, though not as high as the rates for Boston and San Francisco. They are in marked contrast to rates as low as 59 per 100,000 for Detroit and 65 per 100,000 for Charleston.

There are no reasons for believing that the high cancer death rates of St. Paul and Minneapolis are primarily the result of hospital admissions. Probably no city in the country attracts more cancer patients than New York and yet the rate for New York City is only 92 per 100,000. But, granting that there is a liability to serious error, it is obviously of the first importance that the cancer data should be subjected to critical analysis with a due regard to the precise residence of the deceased, the duration of such residence and possible local factors determining cancer occurrence.

The statistics are also suggestive of the urgency of an aroused public interest in *cancer control in the Twin Cities,* carried persistently forward until a measureable reduction in the death rate has been secured. At the present time no other means are known to medical science than the earliest possible removal of the offending mass by whatever means may be considered best. The Society for the Control of Cancer has prepared lecture outlines for such purposes and it is to be hoped that the local committee will gain the proper support from both the medical profession and the laity to carry on its useful work. I may say in behalf of the American Society for the Control of Cancer that it will most readily cooperate in the furtherance of any method aiming at the reduction of the cancer death rate in the two principal cities of Minnesota and possibly other localities in the state likewise afflicted. I may also say in this connection that the society has found the cooperation of the churches most effective in securing a large attendance at meetings. The public is eager for the truth and willing to cooperate when the facts of cancer treatment and cure are made readily intelligible. The society aims in its public meetings to omit scientific terms not within the general understanding, while at the same time all unnecessary harrowing details of the disease are carefully avoided. The expense of local meetings is small, while the results are invariably most gratifying. It is usually a decided advantage to have the meeting addressed by one or two speakers from a distance, to avoid the possible conflict of local interests. Two speakers are generally sufficient for the purpose and a meeting of an hour or an hour-and-a-half meets all reasonable requirements for a full statement of essential facts. The value of the meetings is increased by the use of local mortality or other data illustrating the local incidence of the disease. For this purpose I have for some years past brought together a large amount of statistical information presenting the facts with entire impariality and as derived in a standardized manner from official records. I cannot, of course, on this occasion enlarge upon this aspect of the cancer problem, but I cannot omit to say that the cancer death rate in this country is reaching alarming proportions, corresponding to the highest rates observed anywhere in the civilized world. The tendency of the death rate is so obviously in the wrong direction that I would fail in my own duty if I did not reemphasize the supreme importance on the one hand of more qualified research into cancer causation. and on the other the practical possibility of an effective campaign for cancer control.

I have used the word "*cancer causation*" advisedly. The problem is not one of *a* cause of cancer occurrence, but of many causes and a multiplicity of conditioning circumstances. In my judgment, the research for *a* cancer cause is hopeless, for all the known facts regarding cancer occurrence clearly illustrate the wide diversity of conditions under which cancer occurs with more or less extraordinary frequency. The negative side of the cancer problem is as important as the positive side. It is as urgent that we know or understand why cancer does not occur in certain races, or in certain sections, as why the rate should be one of extraordinary and exceptional frequency. Cancer, for illustration, practically never occurs among our native *Indian tribes,* but I have yet to find the first trustworthy inquiry into the facts of a situaion as puzzling as it gives promise of hopeful results. I have had an analysis made of 161 recent deaths on seven Indian reservations in the state of Min-

nesota, practically limited to the Chippewa tribe. Out of this number of deaths only four were from malignant disease, or 2.5 per cent, which compares with a normal expected proportion of about 4.5 per cent for the population at large. The Chippewas include a considerable proportion, if not a majority, of mixed-bloods. Among full-blood Indians cancer practically never occurs. I have made special inquiries among the Navaos and Zunis, but failed to find a single recorded case. But even more impressive is the result of a recent inquiry by means of a questionnaire sent out to 127 agency physicians, including practically the entire country and a total Indian population of about 115,000. Sixteen out of 71 reported no cases of cancer of the skin, twenty out of 71 reported no cases of cancer of the mouth, twenty-one out of 71 reported no cases of cancer of the breast, and thirteen out of 68 reported no other form of malignant disease. In the remainder of the reports cancer was referred to, in the large majority of cases, as of rare occurrence, and only in very few as of average occurrence, probably chiefly among tribes largely intermixed or intermarried with the white population.

Another most significant phase of the cancer problem, also generally ignored by those who should give qualified consideration to the question, is the very rare occurrence of cancer of the female breast among the *Japanese*. The rate for recent years has been not quite 2 per 100,000 of population, which compares with a rate of 15.7 for the United States registration area and of 20.7 for England and Wales. These rates are for the female population only. They indicate so decided a disparity in disease liability as to emphasize the urgency of a thorough investigation.

Wherever it is possible the facts of local cancer occurrence should not only be brought to public attention, but they should be subjected to a thorough analysis as to possible causative factors. No phase of the problem illustrates this conclusion better than the possible *influence of topography*. The most important contributions on this aspect of cancer control are the statistical studies by Mr. C. E. Green of Edinburgh, who has laid down the fundamental principle that "There are most striking topographical variations in the incidence of the disease" and that when corrected for possible errors due to admissions to local institutions, it will generally be found that towns lying in a hollow, or in a cup formation, have cancer death rates decidedly above the average. Towns on distinctly steep or hilly sites show a cancer mortality slightly above the average, while towns lying on slopes, or on flat sites, or having flat surroundings, show a mortality below the average. These observations by Green, amplified by a mass of strongly convincing information have also failed to attract the required public attention as a new contribution to cancer knowledge. The theories of Green are somewhat involved, since the conclusions are derived partly from the occupational incidence of the disease, partly based on topographic peculiarities and partly on the influence of the fuel, or the relation of cancer occurrence to the elimination and removal of products of combustion from the houses or from the neighborhood. This ingenious theory is based partly on the true nature of chimney-sweep's cancer, in which soot or one of its ingredients is unquestionably the cause of epithelioma. It rests likewise on the theory that coal tar is a causative factor of cancer occurrence among coal tar workers who work with the actual material which induces the disease. It would carry me entirely too far to explain these theories, even in most general terms, but it will be sufficient for the present purpose to have pointed out their importance in the possible solution of the problem of cancer control in the state of Minnesota.

It affords me the utmost pleasure to be able to say that this particular phase of the cancer problem has been made the subject of an exhaustive investigation by *Dr. Georgine Luden* of the Mayo Clinic, who has presented the facts of her findings in a thesis submitted to the University of Minnesota, and which I have had the privilege to read, as well as to discuss with Dr. Luden during my recent visit to Rochester. The work of Dr. Luden is one of extraordinary promise, for the situation in Rochester is apparently precisely such as in the opinion of Green would predispose to an excessive incidence of cancerous disease. The studies by Dr. Luden have not gone far enough and there is the utmost urgency for more extended inquiries, particularly a more thorough analysis of the local mortality experience, inclu-

sive of all the residents of Rochester, since her own investigations are limited to only such as were treated at the Mayo Clinic, but she was very careful to eliminate all cases not strictly representing Rochester residents, who had not lived at least five years in the city. It is practically certain that the investigation will be continued, through the cooperation of the local Board of Health and possibly the State Board of Health, making available the mortality facts for the population as a whole. But the results thus far achieved seem to confirm in a striking manner the conclusion of Green, according to which a smoke-polluted atmosphere is a direct and measurable causative factor in cancer frequency above the normal.

It is to be hoped that further investigations will be made, for it may be questioned whether anywhere the opportunities for *scientific research at a minimum expense* are more conveniently available than at the Mayo Clinic, but Dr. Luden requires the cooperation of various interests representing special aspects of the problem which lie obviously outside of medical and clinical experience. It may be suggested that the facts recently presented by the Fuel Research Board of the Department of Scientific and Industrial Research of Great Britain should be taken into account and that, as far as possible, the method adopted by that Board should be followed. It is also important that there should be the most hearty cooperation on the part of the local Weather Bureau, since the ascertainment of smoke drift and the precise amount of atmospheric pollution are of themselves highly involved scientific questions. I may further refer in this connection to the valuable work on "Smoke; A Study of Town Air", by Cohen and Rushton, London, 1912; to an interesting report on "City Smoke Ordinances and Smoke Abatement", issued by the Bureau of Mines, 1912; the report of the Department of Smoke Inspection of the City of Chicago for the year 1911; and the annual reports and papers of the Coal Smoke Abatement Society of England, as well as the annual reports of the Atmospheric Pollution Commission of Manchester. The latter are unquestionably the most valuable publications of their kind, and as far as practicable, similar inquiries regarding the precise ascertainment as to the degree of atmospheric pollution should be made in the Rochester area. I must also not omit mention of the reports of the Mellon Research Institute, Pittsburgh, although it is most regrettable that the investigations initiated some years ago should not have been continued.

A review of the recent literature and *information on cancer* leads to distinctly disturbing conclusions. Much of what is written is obviously amateurish, and exceedingly reckless public utterances have been made on a question of life and death to a considerable proportion of our people. In a recent medical column of a wellknown daily paper, offering free advice on medical questions, a correspondent is informed, merely on the basis of a superficial statement, that "the lump which has been in the mouth, as long as you can remember, is not cancer; see your physician. If this lump should be removed, he will do so; if not, do not tamper with it. The cure for cancer of the mouth is the very early removal of the tumor, or treatment by radium or x-ray, or both." The answer contains, of course, much that is useful, but the danger is that such advice will lead to neglect to consult a physician promptly. The article bears the inscription "Too Old For Cancer", when as a matter of fact, no adult age is exempt from the liability to cancer, particularly the age period above seventy. In the registration area the cancer death rate of males, ages 70 and over, has increased from 645 per 100,000 in 1909 to 758 in 1918; for females the cancer death rate in old age during the same period has increased from 790 to 902. It is, therefore, entirely misleading to say that anyone is too old for cancer, most of all on the basis of a letter and in ignorance of the actual facts. It, of course, may be assumed that the remark refers to the long duration of the cancerous growth, but the heading itself is misleading as much as the inference drawn from either conclusion.

An admirable illustration of the scientific methods of dealing with the cancer problem is an extremely suggestive address on *Cancer of the Tongue* by Sir D'Arcy Power, delivered before the Royal College of Surgeons, November 14, 1918. From this address I can only quote the following conclusions, deserving of most thoughtful consideration: "It ought to be possible to reduce cancer of the tongue to the subordinate position which it occupied before the 17th century in

man and which it still holds among the domestic animals. At the present time syphilis is more prevalent than it has been for many years and the consumption of tobacco has risen from 7,500,000 pounds in 1914 to 8,500,000 in 1918. Much of this tobacco is smoked in the form of cigarettes, and women now smoke on a much larger scale than they used to. It follows, therefore, that if allowed to continue as they are doing, there will be a large increase in the patients suffering from cancer of the tongue. Such an increase can be prevented by a thorough and systematic treatment of syphilis in its initial stage. For, as has been shown, cancer of the tongue has always increased in frequency during the same years that syphilis has been treated inadequately. Persons who are being treated for syphilis, therefore, should be told never to smoke, not to drink to excess and to pay regular visits to a dentist in order that their teeth may be kept in the best possible state and that dentures they may have to wear should be maintained well-fitting and free from rough edges. The address by Sir D'Arcy Powers is one of the strongest arguments against the theory advanced by those whose conclusions are governed by figures rather than by cancer facts, that the observed statistical increase in the disease is apparent and not real. For, if cancer of the tongue cannot be diagnosed adequately, no type of cancer can; and if, therefore, cancer of the tongue is increasing, it is clearly because of an increased liability to the disease and not because of an increased accuracy in diagnosis.

If facts like the foregoing could be brought clearly and emphatically before the public, much good would unquestionably result. But what is especially needed is a collective investigation illustrating the favorable *results of cancer operations,* with absolute impartiality, for a period of sufficient length. The willingness to submit to operations for appendicitis is primarily because of the clear public understanding that fatality in operations is practically nil, provided the disease is taken care of in the early stage. Likewise, progress in cancer operations will be made as the facts of successful operative results become better known and understood. It would seem that the College of Surgeons should take an active interest in this question, for no organization is better equipped for the purpose. Much more is to be gained by research based upon human experience than by over-emphasizing animal research, where the conclusions may, or may not, be applicable to human beings.

The foregoing observations bring me to the question of *heredity,* in which the conclusions derived from animal experimentation are diametrically opposed to the facts, as far as known, for the human race. Cancer is certainly not inherited in human beings in the same sense as a hereditary predisposition is frequently apparent in tuberculosis. Cancer is now so common a disease of adult life that the occurrence of cancer cases in more than one individual in the same family may safely be looked upon as a true coincidence. The cases of exceptional frequency sometimes reported, even granting the absolute accuracy of the facts, are in the nature of abnormalties, with only a slight bearing upon the practical side of the question. There are no reasons why a cancerous predisposition should not be inherited, or, in other words, why persons living the same kind of lives, in the same kind of environment, subject to about the same external influences, should not suffer likewise from cancerous affections, but no statistical evidence is available to prove that cancer is inherited, in the accepted sense of the term, all the experiments on rats, mice and guinea pigs notwithstanding.

Much is said of *cancer villages,* cancer streets and cancer houses. But all investigations by qualified experts have shown the fallacy of the assumption other than as previously observed, that the general enviroment, the topographic features, or perhaps the occupational pursuits may perdispose to an excessive rate of cancer frequency.

Occupational cancers are a fact and not a theory. Chimney-sweep's cancer is unquestionably a direct result of soot irritation, just as x-ray dermatitis is the direct result of x-ray exposure. Here also an extended discussion would be necessary to illustrate the problem from a practical point of view. X-ray dermatitis, however, may be referred to as evidence of external conditions, within our knowledge and understanding, acting as causative factors in malignant disease and there are no reasons for supposing that the true causative porcesses vary extensively from other forms of cancer than in oc-

cupational skin disease. It may be suggested in this connection that an investigation should be made into the possible occurrence of cancer as the result of employment in coal briquet making while at the same time more knowledge should be forthcoming as regards the possible effect of a smoky atmosphere on cancerous processes.

An important phase of the cancer problem is the higher rate of frequency of *cancer of the uterus among* married than among single women. This aspect of the question has been thoroughly inquired into by the British Registrar General, whose reports on the subject appear to be quite conclusive. The investigation has brought out the further and startling fact that cancer of the breast is relatively more frequent among the unmarried than among the married, and the same holds true of malignant ovarian tumors.

The statement has been made by Deelman, on the basis of Amsterdam statistics, that the average age at death from cancer of the body of the uterus was sixty-one years, but from cancer of the cervix only forty-nine years. Deelman's impressions from statistics, apparently not dealt with as critically as would be necessary, are that unmarried women develop fibromas in the uterus more frequently than the married. The material considered by Deelman and Treub should be reexamined by those qualified to do so. The subject of cancer mortality has for many years received special attention on the part of the Bureau of Municipal Statistics of Amsterdam and few governmental offices could render a more substantial service to the cause of trustworthy statistics, provided the material available is dealt with from both the medical and the statistical viewpoint by those thoroughly qualified to do so. The disturbed population conditions in Great Britain and the long intervening period since the taking of the last census preclude very trustworthy calculations of cancer rates at the present time, but according to the most recent British statistics, for the year 1919, the cancer death rate of women has increased at all ages over 75, while at younger ages a possibly stationary condition seems to have been reached. With reference to marital condition, the report for the period 1911-'13 shows the following significant differences: For cancer of the ovaries the rate was 6.0 per 100,000 for unmarried women, against 3.1 for the married and windowed. For cancer of the uterus the rate was 16.9 per 100,000 for single women, against 29.3 for married women and widows. For cancer of the breast the rate was 34.6 for the unmarried and 23.8 for the married and widowed. This evidence would seem absolutely conclusive, the populations having been standardized and all care having been taken to secure accuracy in the results. The data leave no other conclusion than that unmarried women are more liable to cancer of the ovaries and the breast, while the married are more subject to cancer of the uterus, no details unfortunately being available to determine how far this conclusion applies to cancer of the body of the uterus and the cervix, but there are the strongest reasons for believing that a qualified analysis would bring out equally striking differences.

In this connection, I may again refer to the rarity of cancer of the breast among Japanese women, and as a matter of record I include the latest available data, according to which the rate is 2.0 per 100,000 of female population for the Empire of Japan, as compared or contrasted with a rate of 20.7 for England and Wales, 15.2 for Ireland and 18.2 for Scotland. All of these rates are for the period, 1914-1918. The data for the previous five years support identically the same conclusions. I, however, have gone further into this matter and made special inquiry of practicing physicians in Hawaii, thoroughly familiar with Japanese women, who report that cancer of the breast among them is extremely rare. It certainly would seem worth while that this extraordinary difference should be made the subject of special inquiry on the part of those qualified to do so.

Finally, I may refer to the apparent difference in cancer occurrence according to *economic status*. There seems to be no serious question of doubt but that cancer is more frequent among the well-to-do than among the poor; and since prosperity is practically the equivalent of hyper-nutrition, the conclusion would seem justified that over-nutrition is a predisposing factor of considerable importance. It is true that there is a divergence of opinion on this question, but my investigations conclusively prove that cancer is more common among persons of overweight than among those whose weight falls below the normal standard. Since poverty and under-nutrition, or malnutrition, go practically to-

gether, there is here another neglected field where specialized research gives promise of useful results. Dr. Luden's investigations into the metabolism of cancerous patients add materially to our existing knowledge on this subject.

The foregoing are merely a few random suggestions as to the direction in which cancer research is likely to yield useful results. The subject is one of such a multitude of aspects that there is practically no end to the possibilities of an effective coordination of existing scientific agencies to bring about a better state of knowledge than is extant at the present time. However difficult it may seem, the medical profession owes it to itself to encourage at least such research in two principal directions in which the public has perhaps the most profound, as well as the most practical interest. The first of these is the ascertainment of *after-results of operative interference*, to which I have had occasion to refer previously, but which I wish to emphasize again, as perhaps the most urgent need of a cancer campaign. The second is more difficult, but nevertheless equally urgent, and that is a thorough-going inquiry into so-called *cancer cures*, widely advertised, widely disseminated and, I am afraid, widely used. Whoever has gone into this matter must be familiar with the similarity of assertions, both as to the origin of the alleged cures and the methods of treatment employed, as well as the vague nature of the results. Yet, there are some extremely interesting exceptions to this statement, which indicate possibilities which it would not seem right and proper should be complacently set aside. I have during the present year had occasion several times to concern myself seriously with alleged cures, involving far-reaching possibilities of material harm. I firmly believe that the medical profession owes it to itself, as well as to the public, that it should initiate a thorough-going inquiry into any and all alleged cancer cures, or forms of treatment, not approved by physicians of reputable standing. The British government considered this question in a parliamentary investigation and the recommendations following were as drastic as they were likely to be effective. As an illustration of the dangerous nature of even world-wide propaganda for alleged cures, I may mention a press dispatch from Berlin to the effect that "Cancer has been cured by the injection of child's blood into the veins of a cancer patient." Cases were announced by a doctor of Hamburg, whose name is a matter of record, and although both patients had been near to the point of death, the cure had apparently been suuccessful, two cubic centimeters of blood having been injected twice daily. This astounding dispatch is amplified by the statement that "Cancer never affects children and it is one of the oldest theories in medicine that the fluids of the body have a healing power." It is by such assurances as these that the public is led astray, but happily there is a strong voice of protest from those entitled to be heard.

In conclusion, I wish to reemphasize the urgency of a well-considered educational movement in this state and section, aiming deliberately at the cancer education of the public. By calling public attention to the value of early diagnosis and early operative interference, a vast amount of good can be achieved in the direction of a material lowering of the cancer death rate, which is now not only excessive, but progressively increasing.

REFERENCES

1. Cancer of the Colon by Sir W. Arbuthnot Lane, Lancet, Dec. 11, 1920.
2. Surgical Treatment of Cancer of the Rectum, by W. E. Miles, British Medical Journal, Nov. 13, 1920.
3. The Present Position of Cancer Research, by J. A. Murray, British Medical Journal, Oct. 30, 1920.
4. Pariffinoma and Wax Cancer, by B. F. Davis, Journal American Medical Association, Dec. 18, 1920.
5. The Relation of Cancer to the Prolongation of Human Life, by Wm. J. Mayo, Northwestern Insurance, 1920.
6. The Cancer Igmia, by Robt. Bell, Medical Record, Feb. 28, 1920.
7. The Factor of Fever in the Diagnosis of Cancer, by W. Gordan, Lancet, June 19, 1920.
8. Interim Report of the Special Committee on Cancer, of the Academy of Medicine, Toronto, on Glover Serum; a Glance at Some of the Old and New Theories of the Causation of Cancer, by J. Rawson Pennington, Chicago, 1919.
9. Fads of an Old Physician, by Geo. E. Keith, London, 1897—Chapter VII, on Cancer.
10. Centrollogy, by Rufus Choate, Washington, 1916.
11. Observations on Cholesterin Retention as a Factor in Cell Proliferation, by Georgine Luden, Rochester, Minn.
12. Six Papers, 1916-1919, Journal of Clinical Medicine, St. Louis, Mo.
13. On Changes in the Recorded Mortality from Cancer and their Possible Interpretation, by Major Greenwood and Francis Wood, London, 1814.
14. An Inquiry into the Relation between Social Status and Cancer Mortality, by Brown and Lall, Cambridge, 1914.
15. Bulletin of the Swiss Actuarial Society; Address

On The Mortality from Cancer in Switzerland, 1880-1919, by Dr. J. Aebly, Berne, 1919.
16. Statistical Summary of the Mortality from Cancer In the City of Gall, by Dr. Ernst Jessen, of the Municipal Health Dept. of the Canton of Gall, 1920.
17. Gastro-intestinals and their Sequelae, but Especially Cancer, by Sir Thomas Oliver, London, 1920.
18. Papers on Cancer, 1916-'20, by Dr. L. D. Bristol, State Commissioner of Health, Augusta, Me.
19. Age Distribution and Age Incidence in Five Hundred Cases of Cancer of the Uterus, by Dr. Reuben Peterson, Dec., 1919.
20. On Cancer of the Tongue, by Sir D'Arcy Power, London, 1919.
21. What we Know about Cancer, American Society for the Control of Cancer, July, 1919.

PRESENT STATUS OF SPLENECTOMY AS A THERAPEUTIC MEASURE*

H. Z. Giffin, M. D.
Division of Medicine, Mayo Clinic.
Rochester, Minn.

Introduction

The Southern Minnesota Medical Association is a sectional society with a national outlook. There is a family spirit in its membership which is unique among medical associations. It is large enough to be free from cliques and misunderstandings, yet small enough to maintain a community interest. I am pleased to be its presiding officer. The association is diversified in its personnel. Men who are widely known for their researches, their special skill, or their medical vision come into close contact with private practitioners who have a wide general knowledge of medical practice and in addition to this a special knowledge, which the general practitioner himself does not appreciate, of the early symptoms of disease.

One who has carefully read Sir James McKenzie's recent book entitled "The Future of Medicine", is, I believe, convinced that the future of medicine lies in the realm of the general practitioner, chiefly because of his opportunity to study the very earliest symptoms of disease, to watch the development of disease from its very incipiency, and to ascertain the prognosis of disease as the result of his opportunity to observe patients over periods of years. He must of course, foster the research spirit, learn to investigate accurately, and above all to keep records.

Sir James McKenzie divides disease into four stages:

1. The predisposing stage in which the individual's resistance may be low and he may be susceptible to the attack from without.
2. The early stage in which there is no perceptible alteration of tissue.
3. The advanced stage in which there is definite modification of tissue, and physical signs are present.
4. The final stage, or death.

McKenzie points out that most divisions of knowledge evolve forward while medicine has evolved backward. A vast amount of knowledge has been accumulated concerning the advanced stages of disease but the early periods during which there are no perceptible changes of tissue, and the predisposing elements are those which we must learn to study; and in many instances we do not even know how to attack the problems.

The general practitioner has always had the opportunity to discover those things which we need most to know; that is,the conditions favoring the onset of disease, the circumstances which lead up to the invasion, the early symptoms of disease and their production. The general practitioner has not, however, developed the habit of keeping records nor has he heretofore been educated in the schools for clinical investigation. He has in some instances educated himself and has become especially interested in a study of certain phases of disease. When this has occurred the result has frequently been the discovery of a very important fact.

A medical association of the type of the Southern Minnesota Medical Association should, I believe, make a direct effort to stimulate clinical research among its members. In my own experience I long ago determined that the general practitioner has a great fund of knowledge concerning disease which no one else possesses and which he himself is not in a position to impart. I hope that some plan may be evolved by which members of this association may be referred through the secretary, or some special officer of the society, to those who are able to give them actual assistance in the solution of any problem which they wish to attack.

*President's address before the Southern Minnesota Medical Association, Mankato, November, 1920.

Our programs may then be made representative of the entire field of medicine.

THE FUNCTION OF THE SPLEEN AS RELATED TO CLINICAL EXPERIENCE

The first essential fact with regard to splenectomy is that it eliminates the largest lymphoid organ in the body. That the spleen is structurally lymphoid is frequently overlooked in discussions of splenic function and very little clinical evidence is at hand concerning the effect of splenectomy in conditions associated with lymphocytic hyperplasia. The second outstanding clinical fact with regard to splenectomy is the definite establishment of its remarkable effect in the cure of hemolytic jaundice. It seems most likely that if the spleen has a specific function this function will be discovered in connection with a study of its activity in hemolytic jaundice. The icterus of hemolytic jaundice is largely the result of increased hemolysis and hemolysis seems to be intimately related to the function of the liver. Splenectomy can be said to be a symptomatic cure for hemolytic jaundice unless the anemia has been of such long duration and of such severe type that it has assumed the features of a primary anemia, in which event the blood count does not return to normal, but the patient lives indefinitely. A third striking clinical fact concerning the effect of splenectomy in all forms of disease in which splenomegaly is associated with a secondary type of anemia is that the anemia itself is less severe after splenectomy than it was before, even though the disease may not have been cured by the operation. In the fourth place removal of a very much enlarged spleen markedly reduces the amount of blood which has to pass through the liver. The removal of a small spleen is of doubtful effect on the liver. If a small spleen associated with an advanced portal cirrhosis is removed the clinical result may not be satisfactory. If the large spleen of splenic anemia which has become associated with an equally advanced portal cirrhosis is removed, the clinical result may be quite remarkable, yet there is no reason definitely to assert that the etiology of the two conditions is essentially different. Fifth, it must not be entirely overlooked that the removal of an abnormally large spleen may be a relief to the patient from a mechanical standpoint, although this factor alone would rarely lead to a decision for splenectomy.

SPLENECTOMY

At this time I shall give a general review of the 245 cases in which splenectomy has been performed at the Mayo Clinic up to September 20, 1920 (Table 1).

Table 1
SPLENECTOMY
To September 20, 1920

	Cases
Splenic anemia	71
Septic splenomegaly	10
Hemolytic jaundice	32
Pernicious anemia	53
Myelogenous leukemia	26
Portal cirrhosis	10
Biliary cirrhosis	6
Luetic splenomegaly	6
Lymphocytic splenomegaly	6
Gaucher's disease	4
Tuberculosis of the spleen	4
Wandering spleen	2
Hodgkin's disease	1
Eosinophilia with splenomegaly	1
Neutrophilia with splenomegaly	1
Local bleeding at operation	1
Miscellaneous, questionable diagnosis	11
Total	245

OPERATIVE MORTALITY

The number of hospital deaths in the series of 245 cases was twenty-six (10.6+ per cent). This mortality can not be applied, however, to individual groups of cases. The percentage is somewhat higher in cases of large adherent spleens and in conditions in which hemorrhage is apt to occur, and is very much lower in cases in which the spleen is small and the operation is not accompanied by technical difficulties (Table 2).

Table 2
HOSPITAL MORTALITY IN THE LARGER GROUPS OF SPLENECTOMIZED PATIENTS

	Cases	Deaths	Per cent
Splenic anemia	71	9	12.6
Hemolytic jaundice	32	1	3.1
Pernicious anemia	53	3	5.6
Myelogenous leukemia	26	1	3.8

The hospital mortality for splenic anemia is much higher than for any other group. The spleen of splenic anemia is large and fibrous; perisplenitis is common; the blood vessels are usually markedly enlarged sometimes thrombotic and the operation is correspondingly difficult. Similar conditions occur in septic splenomegaly in which a chronic recurring sepsis over a period of years has resulted in an enlargement of the spleen accompanied by secondary anemia. In

fact the only distinction between splenic anemia and septic splenomegaly is that the process is localized in splenic anemia and the history and the findings are of a more generalized chronic recurrent infection in cases of septic splenomegaly. Much clinical evidence indicates that splenic anemia is a localized form of chronic sepsis. The low operative mortality (3.1 per cent) in cases of hemolytic jaundice is especially gratifying in view of the excellent ultimate result in these cases. There was a similarly low operative mortality in cases of pernicious anemia and myelogenous leukemia.

SPLENIC ANEMIA

Table 3 shows the course of patients splenectomized for splenic anemia.

Table 3
SPLENIC ANEMIA

Cases to September 20, 1920	71
Hospital deaths (12.6 per cent)	9
Subsequent deaths	22
Patients heard from	28
In good condition	22
In fair coindition	5
In poor condition	1
Patients operated on five years or more	27
Patients who lived more than five years	17
Still living	16

Note: One patient is well eleven years and five months after operation.

Immediate hospital mortality was due to hemorrhage in only one instance. Thrombosis of the portal and mesenteric vessels caused death in two instances, while influenza pneumonia, and pulmonary embolus account for the others. The immediate occurrence of localized hematoma, subdiaphragmatic abscess or left pleurisy were important factors in protracted convalescence.

The large number of subsequent deaths (twenty-two) will be noted in this group. The cause of these subsequent deaths at once becomes a matter of inquiry. Four of the patients who recovered from the operation died within three months. Fifteen of those who died later lived longer than two years. The average length of life of those who recovered from operation and who have subsequently died was two years and five months.

It is an important observation that hemorrhage in the form of hematemesis was evidently the cause of death in eight instances. In two of these the fatal hemorrhage followed operation by five years and two months. So far as we know postoperative hemorrhage occurred in a total of fourteen cases. Cirrhosis of the liver, with ascites and severe anemia, is given as the cause of subsequent death in four cases, although it was doubtless a contributive cause in a majority of the deaths.

Definite knowledge that twenty-seven of twenty-eight patients recently heard from are in good or fairly good condition is important and seems entirely to justify the continuance of splenectomy in cases of simple splenomegaly in which the process is localized to the spleen or to the spleen and liver. A demonstration that the splenic enlargement is marked and evidence that the enlargement of the spleen was primary are essential in the decision for surgical treatment in splenic anemia.

SEPTIC SPLENOMEGALY

Although a great deal of evidence indicates that splenic anemia is the result of a localized form of sepsis it seems best to consider separately certain cases of splenomegaly which are associated with a history of chronic recurrent attacks of one form or another or several forms of septic exacerbation; for example, localized phlebitis, recurrent tonsilitis, recurrent furunculosis, or ulcerative endocarditis. In such cases marked enlargement of the spleen, frequently associated with severe anemia of the secondary type and even presenting leukopenia, may eventually develop. In other words in most respects aside from their previous history, they may closely resemble splenic anemia. Table 4 shows the data in ten cases of this type which have been separated from the cases of splenic anemia.

Table 4
SEPTIC SPLENOMEGALY

Cases to September 20, 1920	10
Hospital deaths (20 per cent)	2
Subsequent deaths	5
Patients living	3
In good condition	1
In fair condition	2

The hospital mortality of 20 per cent may be accounted for by the small number of cases in the group, although the mortality would be expected to be slightly higher than in splenic anemia. The fact that five patients have died may be regarded as evidence that the resistance of the organism to disease has been greatly decreased by the chronic recurrent sepsis. The five patients all died within seven months of cardiac or renal disease. Only one of the three

patients living is in good condition. The conclusion seems to be clear that the patient with splenomegaly and anemia, which simulates splenic anemia, associated with a long history of chronic recurring sepsis is rarely benefited by splenectomy, probably because there has been previous damage to the function of vital organs which is irreparable. A careful estimation of cardiorenal sufficiency should be made before operation.

HEMOLYTIC JAUNDICE

Hemolytic jaundice, hemolytic icterus, or acholuric jaundice, is a very clearly defined clinical entity. Whether the cases are familial, congenital, or so-called acquired in type, their clinical manifestations are the same and their characteristics exceptionally well defined, that is, (1) chronic jaundice of many years' duration. (2) periodical exacerbations usually associated with crises consisting of abdominal distress, headache, and fever, (3) the absence of bile in the urine and its presence in the stools, except at the time of crises, (4) the development of gallstone colic later in the history, and (5) the finding of an increased fragility of the erythrocytes in the peripheral circulation. Table 5 shows our experience with hemolytic jaundice:

Table 5
HEMOLYTIC JAUNDICE

Cases to September 20, 1920	32
Hospital deaths (3.1 per cent)	1
Subsequent deaths	3
Patients heard from	26
In good condition	22
In fair condition	4

One patient is well nine years after operation.

The cause of one of the deaths in this group was not given; the second patient developed a peculiar form of gangrenous dermatitis which probably had no relationship to the hemolytic jaundice; while the third recovered entirely from his hemolytic jaundice, but died two years and two months later following an operation elsewhere, for gallstones. The fact that three patients seem to be only in fairly good condition is, I believe, to be expected. Not infrequently patients whose history of hemolytic jaundice goes back to infancy are constitutionally somewhat delicate and neurotic. It is not surprising then to find that splenectomy has not entirely eliminated the constitutional inferiority which some of these patients possess. In general our experience with splenectomy for hemolytic jaundice has been satisfactory; the hospital mortality is low, the patients are rapidly cured of jaundice and anemia, and their subsequent health is the good health of the average person.

PERNICIOUS ANEMIA

Clear indications for splenectomy in cases of pernicious anemia have not been anticipated at any time. It was thought, however, that a prolongation of life might be effected. Table 6 reveals the results in our cases.

Table 6
PERNICIOUS ANEMIA

Cases to September 20, 1920	53
Hospital deaths (5.6 per cent)	3
Subsequent deaths	43
Patients living	7

Eleven patients lived more than three years after operation, five are still living between four and five years after operation.

It will be observed in this table that the hospital mortality is satisfactory, 5.6 per cent. The large number of subsequent deaths in this group is to be expected. It is rather surprising, however, that eleven patients lived more than three years after operation, and that five patients are living in good health and able to work almost five years from the time of operation. That is, 20 per cent of the patients lived more than three years, giving a total duration for the disease of more than four years, while 10 per cent of the patients are living almost five years from the date of operation, giving a total duration of the disease of almost six years. These statistics quite definitely indicate a prolongation of life in a certain percentage of the patients. Whether these findings would be sufficient to lead to splenectomy in many cases of pernicious anemia is questionable; on the other hand, it might seem advisable in individual cases, after all aspects of the question have been considered, to decide on splenectomy. Splenectomy will without doubt cause an increased activity of the bone marrow, and in almost every instance will lead to a remission of at least several months. The operation is certainly not yet to be discarded.*

MYELOGENOUS LEUKEMIA

The patients with myelogenous leukemia were splenectomized after preliminary treatment

*Giffin, H. Z. and Szlapka, T. L.: The treatment of pernicious anemia by splenectomy. Jour. Am. Med. Assn., 1921, lxxvi, 290-295.

with radium. The results are outlined in Table 7.

Table 7
MYELOGENOUS LEUKEMIA

Cases to September 20, 1920	26
Hospital deaths (3.8 per cent)	1
Subsequent deaths	15
Patients heard from	7
In good condition	5
In poor condition	2

One patient died five years four and one-half months after operations; seven patients lived more than three years after operation, six of these are still alive.

The hospital mortality after preliminary treatment and reduction in the size of the spleen is satisfactory, 3.8 per cent. The total number of subsequent deaths in this group is somewhat smaller and the length of life of some of the patients somewhat longer than would be expected; seven patients lived more than three years after operation and six of these are still living. It has been observed that the patients are less likely to develop severe anemia if they have been splenectomized. They are also relieved mechanically of the discomfort associated with splenic enlargement, they are much more likely, however, to have great enlargement of the liver, but this enlargement does not seem to be so great a cause of discomfort as marked enlargement of the spleen. The removal of the spleen in very chronic types of myelogenous leukemia in which the organ is fibrous and the leukocyte count never very high may be definitely advisable. In certain other selected cases, in view of all the circumstances, splenectomy may be recommended, but in the great majority of cases of myelogenous leukemia it is of questionable value.

It is inadvisable at this time to consider in detail all of the remaining groups of cases both because of the variety of conditions included and because of the small number of cases in each group. I shall, however, review in some detail portal cirrhosis, biliary cirrhosis, lymphosarcoma, and benign lymphoma, syphilitic splenomegaly, and localized tuberculosis of the spleen.

PORTAL CIRRHOSIS

It is probable that every case of marked splenomgaly is associated with a certain degree of cirrhosis of the liver. From the pathologic standpoint cirrhosis of the liver may best be grouped as portal or biliary in type. This classification is satisfactory from the clinical standpoint, although a certain number of cases will be quite definitely mixed in type. The cases which I have classified as portal cirrhosis are those in which there seems to be no question that the cirrhosis of the liver is the primary and predominant condition. Cases of splenic anemia in the Banti stage are not included. The diagnosis has been made after a consideration of the clinical, the surgical and, in some instances, the postmortem findings. Occasionally a case is observed, however, in which it is impossible to determine whether the splenomegaly or the cirrhosis is more marked and in which the pathologic change in the spleen and in the liver seem to go hand in hand. Whether there is any essential difference between splenic anemia, septic splenomegaly, Banti's disease, and cirrhosis of the liver remains questionable. Ten cases were grouped as portal cirrhosis. The hospital mortality was very high in this group; four patients died. Three other patients died subsequently within one year of operation. One patient is in fair condition one year following operation, one is living four years following operation, but we have no report of his condition, and another is in good condition two years following operation. Although the number of cases in this group is small it is quite definite, I believe, that in cases of advanced portal cirrhosis in which the spleen is not markedly enlarged splenectomy should be advised with extreme caution. On the other hand in patients with splenic anemia in which a marked splenomegaly has existed for years and portal cirrhosis with ascites has developed later splenectomy may be followed by remarkable results. This differenece is probably due to the fact that splenectomy in the cases in which the spleens were small reduces the amount of blood passing to the liver much less than in cases in which the spleens were very large.

BILIARY CIRRHOSIS

Biliary cirrhosis is as a rule associated with an enlarged smooth liver, early jaundice, and slight or moderate splenomegaly. Frequently there is evidence of increased hemolysis. Six cases have been classified in this group; all of these patients are dead. One patient died in the hospital and five have died subsequently. The length of life after operation has been somewhat longer than in the cases of portal cirrhosis. One patient died five years after operation, one

three years, the remaining three in less than two years.

LYMPHOCYTIC SPLENOMEGALY

In this group are considered the six cases in which the spleen has shown an extreme degree of lymphocytic hyperplasia. There is a distinction between the lymphosarcoma which originates in a definitely localized tumor with subsequent metastasis and the less malignant lymphoma or lymphadenoma which is more generalized. It may be impossible to differentiate even pathologically active malignancy and the more benign processes in the tissue from these cases. One of the patients returned within six months with generalized glandular enlargement and died within one year following operation. The course was that of a malignant condition. One other patient died eight years following operation; the cause of death is not definitely known, but malignant involvement of the liver or cirrhosis is suspected. The remaining four patients are alive and in good condition, one thirteen years, two more than two years, and one six months after operation. The good result following splenectomy in cases showing lymphocytic hyperplasia of the spleen may be of more than incidental importance.

SYPHILITIC SPLENOMEGALY

Our experience with splenectomy in cases of splenomegaly associated with syphilis and syphilitic liver has been very satisfactory. The operation has been advised when antisyphilitic treatment failed to bring about satisfactory improvement in the general condition and in the anemia. Splenectomy was performed in six cases of this type. There was one death in the hospital; the patient died of hemorrhage. The remaining five patients are alive and all seem to be in good condition, two, six years following operation. Treatment for syphilis was more effective following splenectomy than before.

LOCALIZED TUBERCULOSIS OF THE SPLEEN

If it can be demonstrated that tuberculosis is chiefly localized in the spleen or about the spleen splenectomy is followed by very good results. Four tuberculosis spleens occurred in this series. There was one subsequent death of generalized tuberculosis six months following operation. It is probable in this instance that active lesions were present elsewhere in the body at the time of operation. The other three patients are in good condition, one more than three years following operation and two more than four years following operation. A marked anemia was present which promptly improved after splenectomy. Two patients had a previous history of tuberculous peritonitis.

SPLENECTOMY FOR MISCELLANEOUS CONDITIONS

Data concerning the postoperative duration of life in the remaining groups of cases in this series are given in Table 8.

Table 8

SPLENECTOMY—MISCELLANEOUS

Diagnosis	Cases	Hospital deaths	Subsequent deaths	Deaths	Remarks
Gaucher's disease	4	1	1	One more than 3 years after operation.	No report fron one; one in good condition more than 12 years after operation.
Wandering spleen	2	0	0		One in fair condition more than 7 years after operation and one in good condition more than 12 years after operation.
Splenomegaly with eocsinophilia	1	0	1	More than 4 years after operation.	
Splenomegaly with neutrophilia	1	1			
Local bleeding at operation	1	0	1	Less than 1 year after operation.	
Hodgkin's disease	1	0	1	Less than 1 year after operation.	
Miscellaneous questionable	11	2	5	Four less than 1 year and one less than 2 years after operation.	No report from one; one in good condition less than one year and one less than two years; one in fair condition less than five years after operation.

SUMMARY

A general survey, especially with respect to operative death and postoperative duration of life, has been made of 245 cases of splenectomy, for various diseases. Detailed consideration of the different groups of cases must necessarily be undertaken in separate papers.

HEMOLYTIC ICTERUS*

WILLIAM J. MAYO, M. D.
Rochester, Minnesota

In almost every community are persons with chronic jaundice who are otherwise in fair health. Inquiry into the history of such persons develops the fact that the jaundice is not the ordinary type of obstructive jaundice, for it lacks the two cardinal features: clay-colored stools, and bile in the urine. The jaundice is usually of mild degree and is not often accompanied by itching. Think of the picture of jaundice from obstruction by gallstones in the common duct, accompanied by pain, at intervals by chills and fever, and increase of the yellowness, the stools clay-colored and the urine loaded with bile; or again of the painless jaundice from obstruction of the common duct from malignant disease, with clay-colored stools and bile-stained urine, the emaciation and progressive deterioration of general health, and in 84 per cent a distended gallbladder to be felt as a rounded tumor below the margin of the ribs. Obstructive jaundice from any cause should be readily distinguished from hemolytic icterus, badly named.

Early writers spoke of hemolytic icterus as chronic biliousness. Murchison, in 1885, reported chronic jaundice of the acholuric type in several members of a family. He appears to be the first to give a clear description of the familial type, and to call attention to the congenital type. Later the acquired jaundice of the same general description, usually in young adults, was recorded in the literature, and it was noted that the acquired disease as a rule developed more rapidly than the familial and congenital, the consequent anemia was more actively progressive, and death occurred from intercurrent disease at an earlier period than in the other types. Hayem, in 1898, gave a description of hemolytic icterus as a clinical entity. Thayer and Morris, in 1911, published the earliest description in this country of the disease. Chauffard demonstrated that there is increased fragility of the red blood cells in the congenital, familial, and acquired types, an observation of the highest importance in connection with this disease. Sanford has worked out a simple process of estimating red blood-cell fragility which is very useful in the Clinic in making diagnoses since this is a basic condition which does not disappear even after cure of the disease.

While it must be admitted that there are differences to be observed clinically in the congenital, familial, and acquired types of hemolytic icterus it can be asserted quite definitely that they are one and the same disease with essentially the same manifestations. The histories in such cases vary markedly; some persons with the congenital and familial types go through life in a fair degree of health, but the rule is that the disease is progressive and eventuates in a fatal anemia. The patient with hemolytic icterus is subjected to many dangers to which a normal person is not subjected and his life expectancy is seriously menanced, and in the more severe cases a fatal ending may be anticipated. The physical findings of these patients are characteristic. The spleen is definitely enlarged, and as a rule the liver is moderately enlarged. So-called crises occur with exacerbations of the jaundice, in which there are malaise, moderate fever, and sometimes chills with increased tenderness over the spleen and liver. The fact that in all cases of hemolytic icterus the spleen is enlarged suggested even to the earliest observers that the cause is splenic. It is well known that one of the functions of the spleen in the adult is to destroy worn-out red blood corpuscles. There is good reason to believe that the spleen does not wantonly destroy healthy red corpuscles but only those which have deteriorated, that the fragility of the red cells in hemolytic icterus is of extrasplenic origin and this cell defect leads to their destruction in the spleen, and that the enlargement of the spleen might be considered a work hypertrophy. Physiologists in early days estimated that all

*Presented before the Southern Minnesota Medical Association, Mankato, November 29, 1920.

the red cells of the body are destroyed and replaced in the course of a week. This estimation was based an a consideration of the bile pigment. It was believed that all the bile pigment is derived from red-cell destruction. The work of Whipple has shown that there are other sources of origin of bile pigments. Winifred Ashby, by a series of very interesting experiments in the Clinic, has demonstrated that the red blood cells may live from thirty to forty days, and it is quite probable that they may live much longer.

The spleen may be looked on as an adjunct to the liver and its function considered largely as that of a strainer, the material strained being sent to the liver for final elaboration. The enlargement of the liver in hemolytic icterus is possibly also a work hypertrophy, at least the hepatic tissue in our cases showed cell hyperplasia. The liver is exposed to certain secondary changes or complications as the result of being inundated with red-cell pigment, and this is shown by the tendency to the formation of pigment gallstones; such gallstones have been found in three of every five of all cases of hemolytic icterus in which we have operated. The gallstones are a menace and may cause infections in the gallbladder and in the common duct which may travel upward into the liver ducts, producing a complicating biliary cirrhosis as a result of the deposit of connective tissue around the bile ducts; a combination of hemolytic icterus with obstructions from infections of the biliary ducts may interfere with the diagnosis. In several of our early cases the clear-cut history of gallstone attacks, the extension of infection to the liver ducts and to the pancreas, causing chronic pancreatitis, made the gallstones appear to be the primary disease and the splenomegalia secondary. To our surprise after all the stones were removed the jaundice continued, although it was very evident that it was not due to obstruction. The stool and urine had become normal in color and the gallstone attacks had disappeared, although there was a continuation at intervals of the abdominal crises. Later, removal of the spleen cured these patients. Specimens of liver removed for microscopic examination in some of the cases have shown the hyperplasia of the liver cells to be accompanied by evidences of biliary cirrhosis secondary to the gallstone disease.

Hanot's disease of the liver may be a clinical entity. I am not certain that I have ever seen a case. It certainly has no pathologic basis. The fact that it occurs in the young and that the spleen and liver are both enlarged leads to the suspicion that the majority of conditions called Hanot's cirrhosis are either biliary cirrhosis or hemolytic icterus, or a combination of the two.

My attention was first directed to the surgical significance of hemolytic icterus in 1915 by Kanavel's and Elliott's splendid paper. They reported from the literature forty-eight cases in which operation had been performed with but two deaths, thus definitely determining that splenectomy cures the disease. Kanavel and Elliott point out that several successful splenectomies were performed before there was real knowledge of the disease. Spencer Wells, in 1888, and Bland-Sutton, in 1895, had contributed the report of a splenectomy to the literature on the subject. Krumbhaar, in 1915, in a review of 158 cases, showed that 14 per cent were congenital, 35 per cent acquired, and 51 per cent familial. Since the three conditions are alike too much consideration should not be given to the type of the disease, but rather to its effect on the blood, and while it is true that the acquired type is potentially more serious, both congenital and familial types are slowly progressive and may require splenectomy.

Minkowski, Eppinger, and Banti, point out the direct relationship of the spleen to all types of hemolytic icterus.

I know of nothing more remarkable in the results of surgery than the disappearance of jaundice, which the patient may have had all his life, within three or four days after removal of the spleen, and the speed with which recovery from the anemic state takes place. We have removed the spleen in thirty-two cases of hemolytic icterus, with one death, and this death should have been avoided; the spleen was removed during a crisis; in a few hours the patient developed delirium, and died in coma forty-eight hours after the operation. It is very evident that these patients should not be operated on during a crisis.

The technic used in the Clinic for splenectomy

is that developed by Balfour and reported in 1916. A midline incision, as pointed out by Ochsner and Percy, is preferable to any of the left lateral incisions, since it permits access to the gallbladder, appendix, and so forth, and severs few blood-vessels. The spleen in hemolytic icterus as a rule is not very adherent and may readily be separated from its peritoneal attachments. If the spleen is drawn downward and toward the midline, as adhesions are separated by the hand, the splenic space can be packed with a large gauze compress which temporarily and usually permanently checks hemorrhages from separated adhesions.[32] The attachments of the spleen to the stomach through the vasa brevia are separated and tied, and the attachments of the splenic flexure of the colon at the lower pole are separated. The superior edge of the spleen is turnd outward and the tail of the pancreas dissected out from the pancreatic notch in the spleen. The spleen is lifted out of the abdomen, the bloodless peritoneal attachments are quickly separated with the knife, and the pedicle grasped with two forceps, cut, and double-tied. As a rule on removal of the pack the splenic space will be found dry; if not, progressive suturing with catgut can be carried along the upper border of the pancreas, uniting the separated peritoneal structures down to the diaphragm. If the bleeding extends onto the diaphragm care should be taken not to tear a hole into the pericardium or the pleura with the needle, an accident which I had on one occasion, fortunately without harm. On inspiration the diaphragm comes down, and the attachments which contain the bleeding point may be caught quickly with the needle with at least 15 cm. of suture slack in the fingers; this enables the catgut to follow the diaphragm as it ascends in expiration, the needle being caught again when inspiration brings it down.

In conclusion I again emphasize that hemolytic icterus is not an infrequent cause of chronic jaundice, progressive anemia, and early death. Splenectomy is the cure.

BIBLIOGRAPHY

1. Ashby, Winifred: Some data on the range of life of transfused blood corpuscles in persons without idiopathic blood diseases. Med. Clin. North Am. 1919, iii, 783-799.
2. Balfour, D. C.: The technic of splenectomy. Surg., Gynec and Obst., 1916, xxiii, 1-6.
3. Banti, G.: La splenomegalie hemolytique. Semaine med., 1912, xxxii, 265. Abst. Jour. Am. Med. Assn., 1912, lix, 73.
4. Chauffard: Pathogenie de l'ictere congenital de l'adulte. Semaine med., 1907, xxvii, 25-29.
5. Chauffard: Les icteres hemolytiques. Semaine med., 1908, xxviii, 49-52.
6. Elliott, C. A. and Kanavel, A. B.: Splenectomy for haemolytic icterus. Surg., Gynec. and Obst., 1915, xxi, 21-37.
7. Eppinger, H.: Zur Pathologie der Milzfunktion. Berl. klin. Wchnscher. 1913, l, 1509-1512; 1572-1576; 2409-2411.
8. Giffin, H. Z. and Sanford, A. H.: Clinical observations concerning the fragility of erythrocytes. Jour. Lab. and Clin. Med., 1919, iv., 465-478.
9. Hanot, V.: Etude sur une forme de cirrhose hypertrophique du foie (cirrhose hypertrophique avec ictere chronique). Paris, 1875, 155pp.
10. Hayem, G.: Sur une variete particuliere d'ictere chronique: ictere infectieux, chronique splenomegalique. Presse med. 1898, vi, 121-125.
11. Hayem, G.: Nouvelle contribution a l'etude de l'ictere infectieux chronique, splenomegalique. Bull. et men. Soc. med. d. hop. de. Par., 1908, xxv, 122-140.
12. Krumbhaar, E. B.: A classification and analysis of clinical types of splenomegaly accompanied by anemia. Am. Jour. Med. Sc., 1915, cl, 227-245.
13. Masson, J. C.: Exposure in gallbladder surgery. Ann. Surg., 1919, lxix, 423-425.
14. Minkowski: Uber einen hereditarischer unter dem Bilde eines chronischem Icterus mit Urobilinurie, Splenomegalie, und Nerensiderosis verlaufenden Affection. Verhandl. d deutsch. Congf. innere Med., 1900, xviii, 316. Quoted by Elliott and Kanavel.
15. Murchison: Quoted by Krumbhaar.
16. Ochsner, A. J. and Percy, N. M.: A new manual of surgery, civil and military. 1917, Chicago, Cleveland Press, p. 387.
17. Sutton, J.: Three successful splenectomies. Lancet, 1895, ii, 974-975.
18. Thayer, W. S. and Morris, R. S.: Two cases of congenital haemolytic jaundice with splenomegaly. Observations on haemolytic jaundice. Bull. Johns Hopkins Hosp. 1911, xxii, 85-96.
19. Wells, T. S.: Remarks on splenectomy with a report of a successful case. Med.-Chir. Tr., 1888, lxxi, 255-263.
    Case of splenectomy. Lancet, 1888, i, 724.
20. Whipple, G. H. and Hooper, C. W.: Bile pigment metabolism. Am. Jour. Physiol. 1916, xl, 332-348; 349-359; 1917, xlii, 256-263; 264-279; 544-557.

THREE NEUROPSYCHIATRIC NOTES: EPILEPSY, LETHARGIC ENCEPHALITIS AND THE DEMENTIA PRECOX SYNDROME*

C. EUGENE RIGGS, M. D.
Emeritus Professor of Nervous and Mental Diseases, University, of Minnesota.
St. Paul, Minn.

Hughlings Jackson once remarked that there was no such thing as ordinary epilepsy. Experience confirms this statement. One readily recalls patients showing the symptoms of essential epilepsy in whom a month, or possibly years afterwards, optic neuritis and the objective signs of brain tumor became clearly apparent. A patient of mine, a minister, immediately after his Easter sermon was taken with a severe convulsion† The neurological examination was negative, yet within two months the cardinal signs of a brain tumor were manifested. Another, the wife of a physician, was greatly startled by a convulsion; our findings were all negative, nevertheless six months later the symptoms of a brain tumor were clearly marked. Autopsy showed in both patients deeply seated inoperable growths. Dr. James Taylor, London, reports two cases observed at Queen's Square of apparently chronic epilepsy. In one, symptoms characteristically those of essential epilepsy appeared four years, in the other seventeen years, before any signs of a brain lesion were recognizable. Autopsy as in my cases revealed large neoplasms. Twenty years ago I heard Sir Victor Horsley describe a case in which he found an inoperable brain growth which had been treated on the Continent for several years as an idiopathic epilepsy. Epilepsy developing after the third decade is due either to chronic brain disease or it has existed for years as a *petit mal* or nocturnal attacks of so slight a type as to have failed of recognition on the part of the patient or his family, the occurrence of the convulsion causing him to seek medical advice. It must not be overlooked that the secondary or organic epilepsies may respond as favorably to anti-convulsive drugs as those of the essential type, thus adding greatly to the difficulty of diagnosis; the occurrence of a convulsion by no means predicates its existence; only repeated examinations and prolonged opportunity for observation will enable one to arrive at a diagnosis of the real cause of the convulsive seizure. The terms nocturnal epilepsy is unfortunate since it does not indicate a distinct type; it only means that the attacks follow sleep and consequently occur more frequently at night. (Taylor).

Atypical epilepsies are an interesting study. A patient of mine who for years had suffered from attacks of *grand mal* was greatly surprisesd to have these cease and in their stead to be subject to similar periodic seizures in which the symptoms were those of a generalized tremor of the entire body. Mentality was unaffected, the disruptive cortical discharge affecting apparently only the motor cortex. It is well known that dislocations of the shoulder joint may follow a severe convulsion; not only may this fail of recognition, but there is danger of its being misinterpreted as a paralysis often seen after a Jacksonian epilepsy. Spasmophilia, unfortunately, is too frequently confused with epilepsy, sadly to the detriment of the patient. If one remembers that the former occurs usually between seven months and three years, seldom as late as the sixth or eighth year, that it is seen most usually in the spring and fall, that it manifests a trinity of symptoms, namely laryngeal spasm, tetany, and convulsions, and that there is a marked mechanical and electrical irritability of the motor nerves, the differentiation between these two conditions will not be a matter of great difficulty.

The therapy of epilepsy remains in the empirical stage. Unfortunately the tendency to treat the disease rather than the patient still persists. Taylor, whose experiences with these patients at Queen's Square has been exceptionally great, regards bromide* as the sheet anchor of our therapeutics. Children bear the drug well and as a rule they are given too small doses. The individual case should govern the dosage. Many years ago Seguin advised its combination with chloral; thus used it is most efficacious. I remember one boy who was having one hundred and twenty-five attacks of *petit mal* daily. Under this medication he ap-

*Read before the Minnesota State Medical Association, St. Paul, October, 1920.

*Turner of London reports a cure of 23.5 per cent, with a great benefit to another 50 per cent with small doses of bromide alone.

parently made a good recovery; it is four years since he has had an epileptic attack. A decade or more ago Turner of London, recommended the use of Gelineau's dragees, an anti-convulsive preparation composed of arsenic 1-64th grain; bromide of potassium 15 grains; and picrotoxine 1-128th grain. It gives much better results clinically than bromide either singly or in combination. Bromide disagrees with certain patients, increasing the number of seizures and greatly depressing them. Where it has failed, Taylor has found tincture of belladonna effective. In so-called nocturnal epilepsy he advises that tincture of digitalis be added to the bromide. In the majority of epileptics. I have found luminal an excellent substitute for bromide. Therapeutically, it is certainly far more efficient in controlling the seizures. In 1916, Dr. Ball and Dr. Grinker, in their discussion of Dr. Dercum's paper "Epilepsy with Special Reference to Treatment," before the Section of Nervous and Mental Diseases of the American Medical Association, emphasized its value. The dose varies from three-fourths of a grain to one grain three times a day, or it may be given in one dose at night. Intractable cases may require temporarily from six to nine grains in 24 hours. The dosage should be gaged so as to avoid drowsiness. It is a safe, non-habit forming drug and there are really no contraindications to its use. In 100 cases treated by Grinker, only two patients developed rashes, necessitating its discontinuance, and there were no urinary disturbances. Its combination with extract of pituitary body, where there is evidence of its dysfunction, adds markedly to its therapeutic value. Success in the treatment of this disease has never appeared so promising as since this form of therapy has been on trial. From the patient's point of view it is certainly preferable. There is no mortifying acne and the depressing and stupefying effects of the drug are no longer to be dreaded.

LETHARGIC ENCEPHALITIS

As medical men during the past year we have become only too familiar with the symptoms of epidemic lethargic encephalitis. Professor Netter states that there have been more than fifteen hundred cases in Paris and ten thousand in other parts of France. Any attempt to classify it as to type is futile. One can only designate certain outstanding signs, such as lethargy. The simple fact is, as a recent editorial in the British Medical Journal states, there are probably many varieties of virus not yet clearly differentiated, capable of causing a diffuse inflammation of the central nervous system and producing a clinical picture of an acute nervous disease, its symptoms depending on the point of initial localization of the infection. The distinction between these is yet far from absolute, since many of these symptoms are too often the same. In such affections as typhoid fever, acute milliary tuberculosis, meningitis associated with mumps, tubercular meningitis, acute choreic symptoms, and syphilis of the central nervous system, a differentiation from lethargic encephalitis is a matter of great difficulty. The important fact is that there is a form of encephalitis with a definite pathology giving rise to a clinical syndrome which possesses a great variety of symptoms common to all forms of encephalitis, yet showing certain distinct characteristic features such as lethargy with diplopia, its cause being a virus separate and distinct from that of influenza, poliomyelitis or any of the infections involving the central nervous system. The following classification is suggestive, but is by no means all-inclusive: the paralysis agitans syndrome; delirium with myoclonic contractions; meningeal irritation associated with irritation or paralysis of the cranial or peripheral nerve roots; and hemiplegia with hemianesthesia.

Emphasis should be placed on the early diagnosis of abortive or ambulant cases which too commonly escape recognition. Patients affected with so grave a disease as inflammation of the brain should be advised against the continuing of their normal activities even in the absence of fever, vomiting or headache. Taylor reports a case in which for a time there was on overpowering drowsiness, the patient having to be awakened to take food; after this passed the only symptoms were diplopia, nystagmus and weakness; recovery was rapid. Buzzard and Greenfield call attention to the inclination to fall asleep while sitting down to rest, a tendency to forget the happenings of the day, with possibly a change of temperament and a transient diplopia as the only symptoms. The occurrence of either diplopia or blurring of vision

without an ascertainable cause should be carefully investigated. There are cases in which, athough a partial recovery occurs, yet the patient is greatly changed, and in those manifesting the paralysis agitans syndrome this change would appear to be permanent. In a case recently examined the distinction between it and Parkinson's disease could not be made without reference to the history of the illness. There was an absence of tremor which when present lacks the rhythmical character of this affection. Muscular rigidity was marked, as was also the characteristic posture, the wooden gait, the mask-like expression, the monotonous voice, the everted lower lip, and antepulsion and retropulsion. Mentality seemed unaffected. The future of these patients is one of helplessness and invalidism. The following case would seem to indicate the existence of a chronic form of the disease:

O:, 44 years of age. Father died at 52 of pneumonia; mother is living in her seventieth year and is well except for glaucoma; two brothers and three sisters living and well, one brother died in France. He had the usual childhood infections and 19 years ago contracted lues. Since mild secondary symptoms there have been no indications of the disease. In January, 1920 had an attack of influenza; was in bed ten days during which time his temperature did not exceed 101 degrees, was delirious for several nights. There was headache with double vision for two or three weeks afterwards and he was very drowsy and lethargic. While this patient never regained his normal health, yet it was not until early in March that he noticed he was losing strength. Incoordination of the right arm and leg developed; there was also a right facial palsy which has since disappeared; the least exertion exhausted him; slurring of speech was of late occurrence. His most distressing symptoms have been weakness and vertigo There was Rombergism and Babinski in both feet. Lumbar puncture showed normal pressure, 8 cells, negative Nonne, negative Wassermann both of blood and spinal fluid and a colloidal gold curve 1233431000; urine negative, blood pressure 128 systolic and 80 diastolic, hemaglobin 86 per cent, red blood cells 4,520,000, white blood cells 7,200. On May 25 the neurological examination was negative, aside from a questionable Babinski in right foot. The history of the illness taken in connection with the serological findings indicates clearly lethargic encephalitis.

Diagnosis, especially of the abortive and ambulant types is a matter of great importance. The cardinal symptoms are somnolence, cranial nerve palsies and fever. The significance of lymphocytosis has been much discussed. It is generally accepted that it may occur, but it is the exception rather than the rule, and its absence does not invalidate the diagnosis. Marie believes that a moderate degree of lymphocytosis does not exclude the possibility of encephalitis, but if it should be marked or persistent the question would arise as to whether we were not dealing with a meningitis either of the tuberculous or syphilitic form.

The treatment of lethargic encephalitis with our present knowledge is of necessity empirical. Netter says that while benefit may result from the intraspinous injection of serum taken from a recovered patient, there is no evidence that an antibody appears at an early date in the blood, hence he does not advise this procedure. He insists that urotropin should be given by mouth so as to produce a continuous action on the nerve centres, which does not occur when injected intra-venously. Pilocarpin assists elimination of the virus, but it should be used in conjunction with adrenalin to counteract its depressing effect on the heart. He warns against the use of arsenic, having observed disastrous results following the employment of neosalvarsan. The treatment he has found most efficacious is the creation artifically of an abscess by the injection of from 15 to 30 minims of turpentine into the outer side of the thigh. The beneficial results he attributes to the stimulated activity of the bone marrow. Twenty-five years ago I found Dr. Bruce of Morningside Asylum, Edinburgh, using this method in intractable cases of acute mania. The employment of the nucleinates after this fashion has been for many years a routine procedure with me in manic-depressive insanity and dementia precox. Cases in which a high leucycotosis occurred offered a much more favorable prognosis than those in which there was no reaction. Dr. Bond has observed improvement after dental attention, thyroid therapy, and lumbar puncture. In none of my cases have I observed improvement fol-

lowing the use of urotropin. The only thing that has given a positive result is lumbar puncture repeated as frequently as the necessities of the case seemed to demand.

THE DEMENTIA PRECOX SYNDTOME

The adolescent insanities (manic-depressive insanity, paranoia, and dementia precox) are a malevolent triumvirate. Perhaps of these the syndrome of dementia precox is the least clearly defined and the most chaotic. A decade ago paranoia was believed to be a relatively common psychosis; careful observation, however, has shown us that it was not paranoia but the paranoid forms of dementia precox which were of frequent occurrence. Paranoia simplex, a definite clinical entity, manfests no hallucinations and is of rare occurrence. A distinguishing characteristic of manic-depressive insanity is a recovery from the individual attack with a diabolic tendency to recurrence. A fundamentally defective mental mechanism, a failure in biologic adjustment is basic in all these psychoses, the initial fault being most marked in paranoia and dementia precox. Wolfshon reports finding an hereditary factor of 90 per cent in the latter. White believes it plays an uncertain role. There are also evidences of morphologic deviation. While these insanities were not unknown to Pinel and Esquirol, Morel first used the expression precocious dementia. It was Pick, however, who introduced its Latin equivalent dementia precox, which clouds rather than clarifies its meaning.* The term precox is unfortunate, but it no longer misleads if one accepts White's interpretation that it is precocity of dementia, not a precocity of age. The syndrome is likewise unfortunate containing as it does many symptoms common to the insanities in general.

Hallucinations, especially of the sense of smell (Savage) and loss of memory notably for recent events are common in the senile psychoses. Katatonia is seen not only in juvenile insanity but in organic conditions as well. Bond reports the occurrence of katatonic symptoms in epidemic encephalitis. Last winter an involutional psychosis under my care developed a katatonic stupor which subsequent observation showed to have been due to an encephalitis. The usual classifications of demetia precox are academic rather than real. They are arbitrary, confusing, and unnecessary. The symptoms vary greatly, depending upon the time when the mental mechanism is involved in the course of its evolution. Thus those of the pubescent period are quite distinctive. The waves of depression and expansion are less marked, the delusions transitory, fragmentary and changeable, indicative of the patient's age. While those occurring during the process of and at the close of adolescence are in like manner characteristic, katatonia suggesting early or mid-adolescence, systematized delusions its close. Mixed forms occur in which symptoms common to all may be observed. Striking abnormalities of character and conduct at variance with the social order may be the results of the disintegrating influence in an abortive precox on the mental mechanism, perverting and crippling the higher faculties, thus evolving the radical, the parlor Bolshevist, the crank, the hobo, and the Magdalene. Prognosis is determined by the syndrome and the date of the development of the disease. Systematized delusions, if present, are of ill omen, recovery from the first attack may occur, but its duration is indefinite, recurrence follows and there is a steadily increasing dementia. Age possesses a real significance. Purbescence, because the mental mechanism is in the process of making, is most to be feared. Adolescence is more favorable, and the adult life is distinctly so. Recovery is at the minimum in pubescence (8 per cent); greater in adolescence (13 per cent. Kraepelin), and in the adult it may exceed 50 per cent. The British Medical Journal was moved to protest by the astonishing report of Dr. Beaton of the Royal Navy that he had observed 50 per cent of recoveries in dementia precox. If by recovery one means the getting over an attack occurring in late adolescence or adult life in which systematized delusions were wanting. Dr. Beaton's percentage of recoveries was far too small. Experience has taught me to expect this in a majority of these patients after an illness of four to eight months' duration. The clinical syndrome is quite characteristic. The attack may occur without warning; on the contrary it may masquerade as a manic- depressive

*The same criticism applies to Bleuler's term schizophrenia which indicates a splitting or cleavage of personality and since this occurs in other forms of insanity as well as in the neuroses Dercum believes it should be rejected.

insanity for months or years, before the real nature of the illness is recognized. There may be a preceding insomnia, depression, irritability, erratic conduct, inability of adjustment; elation may alternate or commingle with depression. Mental confusion may be very marked. The delusions are agonizing in character, the hallucinations terrifying, impulsive acts common, and suicidal and homicidal tendencies are almost invariably present. Stupor, catalepsy, posturing, mutism, negativism, grimacing, verbigeration and stereotypy are among the cardinal symptoms. The more acute the attack, the less the probability of mental deterioration. I have known patients to scream out definite sentences at the top of their voices for five consecutive nights and days with the regularity of a fog horn. The stupor is only apparent, not real. After a stuperous attack these patients will describe in minute detail every little happening. One of my cases portrayed in a most amusing manner his observations during a stupor, of the the Romeo and Juliet scenes as acted by the male and female nurses in charge of his case. It is not an attractive picture, yet out of this physical and mental chaos health and sanity may emerge.

The following case illustrates not only the character but also the toxic origin of the precox syndrome: Mrs. S. 51 years of age, mother died of cancer; father of paralysis; a sister insane, one brother and one sister living and well. Two sons both well. Never ill until two years ago when she lost weight, became irritable, and very nervous. September, 1918, the superior thyroid artery was ligated for hyperthyroidism; March, 1919, was operated on for goitre. Two months later she became suspicious, sleepless and acted peculiarly. Delusions variable, changeable, and unsystematized developed. She was acutely excited, noisy, mentally confused, refused her food and was untidy. She was subject to attacks of weeping and depression. Her hallucinations of sight and hearing were terrifying, especially those of hearing. There was posturing, grimacing, senseless laughter and mutism. Hemoglobin 65 per cent, red blood cells 4,050,000; white blood cells 7,200; blood pressure 132-78; pulse 150. Urine contained albumin and white blood cells, no casts. She recovered completely after six months' illness, gaining 23 pounds in weight. The metabolism test was +10.

Evidently the endocrine disturbance was the active factor in this patient's illness. Dana believes that gland defect is sometimes present in the psychoses. Kraepelin suggests a toxic origin. The investigation of Mott would indicate that its source was the sexual glands. Enlargement of the thyroid gland occurs with suggestive frequency in dementia precox (Dercum). Infections and cerebral trauma play an etiological role. An illustration of the former is seen in Mrs. V. a young married woman 31 years of age, always nervous and inclined to worry; except for two confinements had never been sick. December, 1919, she had an appendectomy and a cystic ovary removed. Operation for an infected gall bladder was deferred because of excessive nervousness. Twelve days later she became depressed, irritable, sleepless, and was given to spells of silly laughter. It was at this time I saw her. There were four abscessed teeth and septic tonsils; grave sepsis occurred along with septic arthritis, most of the joints being involved. The psychosis which was distinctly that of a precox type ran a stormy course. She made an excellent mental recovery after an illness of eight months' duration. The dementia precox syndrome was a common form of the psychosis that occurred so frequently after influenza in the never-to-be-forgotten year of 1918-1919. An instance of cerebral trauma* is that of a Montana ranchman whom I saw five years ago. His team ran away, he was thrown to the ground and a wheel of the wagon ran over his head. After the immediate effects of the trauma had disappeared he developed a typical clinical picture of dementia precox.

An interesting feature of these cases is that after months of seemingly hopeless illness *all* symptoms may disappear with a startling suddenness, the patient being able to return home within a week or ten days, apparently perfectly well.

As in the preceding conditions, treatment is empirical. All sources of infection should be removed. More than twenty years ago Sir Thomas Clouston advised the use of the thyroid

*Instead of traumatism being the indirect cause some would regard this as a case of latent dementia precox liberated by the cranial injury.

gland in insanity. The administration of the extract of certain ductless glands or of the mixed glands is a routine procedure. That of the pituitary body has seemed to me to have a distinct value. Injection of the nucleinates is worthy of trial.

Dementia precox, like hysteria (Lasague), is a waste basket in which are cast all the psychoses of which we are unable to make the diagnosis; not only does it include those individuals in whom the initial life impulse, being defective, fails to attain Nature's expectancy, to round out a normal life cycle, but includes as well a wide range of cases not found in our classic conception of the disease, thus offering a more hopeful prognosis than was ever conceived of by Morel and Kraepelin. Hallucinations, loss of recent memories and katatonia can no longer be regarded as distinctive symptoms. In many instances, the delusions, hallucinations and mental confusion are indicative of a toxic psychosis rather than a precocious dementia with a background of neuro-degenerative taint, or a failure in the evolution of the organism with glandular defect and a faulty metabolism.

Both in the psychoses and the neuropyschoses the primary weakness lies in the neural mechanism. Under certain conditions, which include environment, emotional stress, trauma, glandular defect, infections, etc., the resultant reaction may be on the one hand either a psychosis degenerative in character, such as paranoia or dementia precox, or possibly one more benign in nature with an ever-wavering mental balance as in manic-depressive insanity, or perhaps a psychosis temporary in duration and favorable of type, or, on the other, it may be a neuropsychosis. Symptomatologies in insanity are protean in nature, inextricably interwoven and sadly lacking in distinctive characteristics. No Ariadne thread guides us through their confusing labyrinths. Syndromes are tentative merely, not pathognomic. Only in this way can we satisfactorily explain the remarkable results reported by Dr. Beaton which my experience confirms but finds much too small.

DISCUSSION

DR. C. R. BALL, St. Paul: We are greatly indebted to Dr. Riggs for presenting so well these three very important subjets in neurology. I am particularly glad he has called the attention of the members of this section to the use of luminal in the treatment of epilepsy. About nine years ago, after reading the report of a number of cases of epilepsy treated by means of luminal in the Muenchr. Wochenschrift I began to make use of this drug in my epileptic patients. In my opinion luminal gives the best results in epilepsy of any of the remedies which I have prescribed. It also has the advantage of being freer from deleterious results than the bromides. Rarely one sees a dermatitis as a result of the administration of luminal but this may be said to be of very rare occurrence indeed, especially in the small doses which are used in the treatment of epileptic patients. I have given luminal in doses of one and two grains three times a day to the same patient over a period of years and then suddenly taken this patient off from the luminal without noticing any of the nervous disturbances such as follow the sudden withdrawal of habit-forming drugs. From my experience with it, I do not think patients are in any more danger of becoming luminal addicts than they are of acquiring a bromide habit. My observation in patients taking both of these drugs is, the longer they take them, the more pleased they seem when they are discontinued just as patients usually are at the discontinuance of any other medication.

So we may feel that in the use of luminal in the treatment of epilepsy we have a very valuable new remedy which can be safely and effectively administered to our patients over long periods of time.

In the three subjects, epilepsy, encaphalitis and dementia precox, we do not detect a similarity in nomenclature, but if we consider their symptomatology and especially their pathogenesis, their relationship to each other is clearer. This is particularly true of epilepsy and dementia precox. They are enrolled under the same great grouping of the psychoneuroses, the great majority of cases occurring in persons heavily burdened with a bad nervous inheritance. Delusions, hallucinations and a demented state are often terminal conditions and both are quite prone to making their appearance at or about the age of puberty when the sex glands are taking their place as active factors in the endocrine system.

Dr. Riggs has well said that the pathogenesis of these two diseases,—epilepsy of the idiopathic character and dementia precox, is a disturbance of the normal mechanism. I think he would be justified at the present time in going a step farther and saying that this mechanism to which he refers is to be found primarily in the sympathetic-endocrine system.

Just a word in regard to the analogy of the encephalitis cases: Many of them after the passing of the acute stage are left in a condition which bears a striking similarity to the catatonic state so often occurring in dementia precox. Is this catatonic state entirely due to the effects of the encephalitis or has the toxin which produced the encephalitis involved also elements in the sympathetic-endocrine system? Considering the close association of the brain and its membranes to the pituitary gland, I think the latter supposition a likely one.

DR. S. MARX WHITE, Minneapolis: The three things Dr. Riggs discusses in his paper have another thing in common, and that is our almost total lack of knowledge of gross and microscopic pathology and particularly of pathologic physiology. It is for that reason that they must be discussed so largely from the functional side. However, concerning one of them, the present methods of deadhouse (how I hate that term) pathology are bringing out a great deal of knowledge as a basis for certain of the manifestations, and there is at this point one very important thing that I think can possibly be aborted by an increase in our deadhouse pathology. We are today, I think, without even a clue as to whether these forms of encephalitis that we have been seeing in epidemic form in groups, are the same condition that we see sporadically and have been seeing sporadically for a good many years. We have not enough knowledge of these interepidemic forms to say whether we are dealing with the same thing or whether we have a different condition, but there are sporadic cases of what newspapers call "sleeping sickness", individual cranial palsies, or some closely related groups with palsies, and in all of that group it is important to learn whether the pathology of these—to speak nothing of the etiology, of which we know so little—is a common one. For that reason I would like to repeat what I repeat very often, and that is a plea to the general practitioner who sees most of these cases to insist upon the opportunity for autopsy in all of these aberrant encephalitis types and the interepidemic group. In the encephalitic group one of the striking things is the number of cases with aberrant symptoms, during the epidemic period the number of cases we see with a single palsy. Another group that interests me because of the fact that we are dealing with encephalitis, in which we have no lethargy but active mental symptoms or sometimes delirium,—is those rare cases without mental symptoms with cranial palsies. I want to emphasize one particular thing in that group, and that is a prolonged rest. I think that just as in the cases of severe cranial injuries, concussion or fractures, it is necessary to emphasize the need for long rest periods. That period should be too long rather than too short. It is common to allow these patients, as soon as the symptoms have disappeared to resume active life and I have seen cases of relapse as a result of ths procedure. Brain tissue, as we all know, recovers with extreme slowness, and it is essential for complete recovery that a long period of rest be given. I have seen some temporary improvement after the procedure Dr. Riggs mentioned of lumbar puncture, but I have never seen any permanent improvement afterward and doubt if such a procedure could have any permanent influence on the condition.

DR. T. L. BIRNBERG, St. Paul: On looking over this program I see that "bromides are in the discard", and I rise on account of that. It certainly makes me feel badly to have a brain child discarded that had served us so long and so faithfully. However, before we discarded this brain child and make a step-child of it, and put another child in its place, let us put a little more thought on the subject. By bromides we mean a combination of bromine with some other substance, as sodium, potassium, calcium, or magnesium. This gives us a combination of many kinds. If we do not get results from one, oftentimes we try the others. Of course, my remarks are from a very limited field,—the diseases of children,—and so you must take that into consideration. However, when you combine drugs like sodium or potassium with bromine, you are making a mistake, as sodium and potassium are stimulants, and when we combine a stimulant with a sedative, we immediately defeat our purposes, at least in a measure. Calcium and magnesium salts are sedatives, so we should try to combine two sedatives,—calcium and bromine, and then we have a drug that has served us for many years and can be expected to give results.

The word epilepsy is simply a word that means nothing from a diagnostic point of view. It is a symptom that may include many different diseases of different origins. In children, epilepsy so-called, is usually a metabolic condition and in the treatment we try first, if we have sufficient skill, to find the cause. We treat the case and get results from treating the cause and not the symptom; any doctor who cures epilepsy with bromides is the same kind of a doctor who treats arthritis with aspirin. There are two kinds of doctors: those who think and those who dispense pills (laughter), and so in using the calcium bromide we simply use something to help us along while we are proceeding to find the real cause of our trouble. Calcium bromide has been a faithful child to us. There are a few things about calcium and about the bromides,—a few of which I will mention. Children tolerate them very nicely and they almost never have a bromide rash in infancy or childhood. I never knew why this was until I was told that the bromides only cause acne in adults because the sebaceous glands, during certain periods of adult life, are not functioning properly, and so now we can understand why in adult life we have so much acne from the bromides and so little of it in infancy and childhood.

Another factor about our friend bromide is that its action can be greatly assisted by proper diet. You must remember that if you put a patient on a diet without salt, or with very little salt, a small dose of bromide will go a long way,—therefore, in using bromides we should use a salt-poor diet.

A third factor is rest. This thing was worked out very nicely in the Pirquet clinic. They kept a child with epilepsy under observation and recorded the number of convulsions. Then they put the child to bed and found that with the same drugs and other therapeutic measures and diet—but with rest—the number of convulsions was much less.

And so I wish to add to the assistance of our brain child,—the bromides, rest and a salt-free diet.

Another factor to be remembered is that there are

other sedatives, magnesium and calcium salts. How can we diminish the irritability of our nervous system? Giving some lime or magnesium by mouth might help the druggist but would not help our patient very much. There is plenty of magnesium and calcium in all the food we eat, if we could absorb it: We do not absorb it, and, therefore, we should do something to help the patient to absorb the calcium salts supplied by the food. We have a method of assisting the absorption of calcium in the tissues. That is by administering phosphorous dissolved in cod liver oil.

DR. C. EUGENE RIGGS, St. Paul, (closing the discussion): The purpose of my paper was eminently practical. We all treat epilepsy; its therapy is not confined to my specality. As far as Dr. Birnberg is concerned, he is perfectly right in his defense of the bromides. I do not think bromide should be placed in the discard, but I think we have a substitute in a majority of the cases which is ever so much more effective.

As to encephalitis, those of you who were overseas know what it was over there. Those of us here trying to do our work and your work and help bear your burden, know what we passed through here, and I tried to give a resume of the best method of therapy at the present time. I certainly believe that the lumbar puncture is far superior to anything else we have today. I think it takes out the toxic materials along with the spinal fluid and at the same time flushes the canal with antibodies which have a certain therapeutic value.

You know only too well, when you see a patient has dementia precox, it means a practically hopeless prognosis. What I wish to impress upon you is that there is a class of symptoms that is included in the syndrome of dementia precox that most likely is a form of toxic insanity and it does not have the hopeless outlook nor stigma of this unfortunate disease. Fifty per cent or more of these patients recover.

FACTITIOUS TOXICITY OF PREVIOUSLY SIMPLE GOITRES*

From the Material of the Duluth Clinic

T. L. CHAPMAN, M. D.
Duluth, Minn.

For centuries previous to the discovery of iodine and its identification as an element in 1912, there had been used in the treatment of various forms of goitre, throughout the known world, certain marine derivatives such as powdered and burnt sponge, calcined corals, and "aethiops vegitalis," all of which contained iodine in appreciable amounts. In spite of the short waves of popularity of other forms of treatment, such as the application of toads to the swelling, the various charms andd incantations directed against goitre, and even that universal panacea the King's Touch, administration of some form of iodine containued, throughout the ages, to be the most successful of all methods of curing struma.

With the introduction of the numerous salts and solutions of metallic iodine, impetus was given to this treatment, because of the availability of the medicine and its occasional magical results, and this popularity increased to a point where both physicians and laymen were familiar with its curative power. Until comparatively recently every type of goitre in certain parts of this country has been treated at some time with iodine, and this course has been recognized as correct practice by many text books. In fact the summing up of the medical treatment of goitre, as held by a great proportion of practitioners, can be said to have been the use of some form of iodine.

Since the publication of the remarkable studies of pathologic thyroids made by Wilson, much light has been thrown upon the possibilities of harm, under some conditions, by this type of treatment, and it is the purpose of this paper to call attention to the continuing prevalence of a careless disregard for the immensely important effects of this drug, when given without an understanding of what may be grave disaster to the patient.

The excellent results of iodine medication in the simple types of goitre, more especially the adolescent and pre-adolescent varieties, have been attested for years, and it is probable that here the drug has justified the general confidence in it. If evidence to this effect was formerly based upon unscientific and empiric grounds, the late researches of Marine and Kimball have definitely proven that iodine, in the form of one of its salts and probably in many of them, will not only cure but, to a large extent, prevent goitre of these types.

In view however of the fact that, after the adolescent period, a thyroid enlargement, even without symptoms, may be, nevertheless, one that contains the essentials of toxicity, it is well to consider what forces may produce this untoward result. In the first place it is not to b

*Presented before the Minnesota State Medical Association, St. Paul, October, 1920.

forgotten that even in the supposedly simple forms of struma, many observers have noted areas, scattered and relatively small, throughout the growth, that showed evidences of hyperfunction, in that the epithelial lining of the acini was reduplicated, the vessels tortuous and thin walled, and also with more or less papillary formation in the alveoli;—and yet withal these goitres have given no symptoms of toxicity. These findings bear out the statement of McCarrison, who remarks that there are few human beings living under modern conditions of life, in whom the thyroid is wholly normal. It seems also established that animals of several kinds, are also subject to wide variations in degrees of thyroid hyperplasias, especially those creatures confined in limited spaces and in close association with man. It is more than possible that the active hyperplastic areas that the pathologist observes are insufficient in size and bulk to produce a toxicity sufficient to give rise to clinical symptoms of a known type, and it may also be that any small excess dosage of thyroid hormones from these areas, can be neutralized by the balancing and protective forces of other glandular structures of the body providing no unbalancing agent is introduced.

The remark of Ewing[4] is particularly pertinent in this connection. He says "An important principle governing pathological processes in the thyroid, is the remarkable response to functional stimulus, and the wide variations in functional activity and structure of the gland in the different physiological states. Marked cellular hyperplasia may readily be induced by changes in diet and by drugs." It is conceded that certain investigators have declared that the administration of iodine causes a "resting state" of the gland, but our clinical experience certainly does not bear this out as a characteristic result. On the contrary we have seen repeatedly, symptomless goitres hasten to a well defined toxicity, and already toxic cases both partially hyperplastic, and the adenomatous, made more toxic by this drug.

In our present state of knowledge, no amount of clinical acumen will enable us to determine the existence or extent of these excitable areas in goitres that have given no symptoms of hypersecretion; no accuracy of history-taking will enable one to have knowledge of a long forgotten previous activity of the thyroid. If a patient presents himself without notable increase in the basal metabolism, without tremor, tachycardia, sweating hands, loss of weight and strength, digestive and intestinal crises, the diagnosis of simple goitre is made and too commonly iodine is administered. Such a course certainly does not take into account the above mentioned pathological facts, and may precipitate a period of grave danger to a patient.

From the available literature on the action of iodine upon the thyroid gland, Crotti[5] considers that two facts are probable, first that iodine activates the functional activity of the epithelium, and that it also accelerates liquifaction of the colloid. It also seems fair to include with this statement that certainly this drug does hasten absorbtive processes in general. What worse drug could possibly be given in a condition where the pathologic condition of the gland cannot be accurately determined before hand? Aside from the possible presence of the small hyperplastic areas that are admittedly of fairly frequent occurrence in clinically inactive goitres, there is besides the very common adenoma of the thyroid, giving no symptoms for many years after the enlargement of the thyroid is noted, but potentially toxic. Can the encouragement to rapid absorption from these areas, and the early instead of the late, instigation to degeneration in other organs, be considered anything but the gravest of mismanagement?

It has been long known, but partly forgotten, that in some instances toxicity was rapidly produced by this agent. The literature abounds with dramatic examples. It was the similarity in action of thyroid extract and iodine that led Kocher in 1895, to suggest that the extract of the gland might be found to contain iodine, which all the world now knows to be a fact. Roos, in 1899 showed that there was a direct relation between iodine content and the effect of thyroid on metabolic processes, and Halstead's experiments seem to confirm the opinion that iodine either supplies or stimulates increased output of the gland substance. Breuer, quoted by Crotti, called the condition of toxicity caused by iodine the "iodine-Basedow," since the effects were usually those of exophthalmic goitre, headache, irritability nausea, vomiting and later delirium. Kocher[3] as long ago as 1916 called

attention to a rapidly occurring hyperthroidism, occurring in cases of simple goitre treated by iodine, and Crotti again in the last edition of his book says, "Every surgeon who has had some experience in goitre surgery has seen, I am sure, more than once, these artificially produced cases of Graves disease, caused simply by an untimely, exaggerated, and unintelligent treatment with iodine or its compounds."

If these authorities had substituted for the expressions "Graves disease" and "Iodin-Basedow'" that of "thyrotoxicosis" we could find our opinion in entire accord with theirs. For it is a fact easily demonstrable beneath the microscope that these glands do not present the general and widespread hyperplasias and hyperthrophies that are necessary corollaries to the diagnosis of Graves disease according to our best pathologists; nor do these patients ever, as far as the author is aware, show a true exoptthalmos, however much in other clinical expressions they may be similar.

Excluding the quiescent and abortive forms, and the commonly known comparatively inactive periods of true exophthalmic goitre, it seems certain from these facts that it is possible to secure from small hyperplastic areas of a gland, treated by iodine, the quantitative toxicity that resembles a true Graves disease. It is equally certain however, that though these results may be achieved in an extreme grade, that grade does not, in our experience, reach the limits of toxicity or the quality of that disorder, (Graves disease) in all regards. That is, while tremor, loss of weight and strength, tachycardia, and nervousness may be severe in each condition. the author has not seen in any case of activation by iodine, the production of an exophthalmos, excepting where there might be a reasonable assumption that the goitre had been a true exophthalmic one, in some stage or other before treatment had been instituted by the drug. Our findings in the laboratory also confirm this view of the toxicity produced. In no case of previously apparently simple goitres, in which toxicity appeared after the use of iodine, was there found the characteristic general changes in the gland described by Wilson as necessary, before the case can be considered a true Graves disease. These glands were generally larger than the true Basedow gland, by gross examination not so dry or granular throughout, and under the microscope the changes in the cellular elements were confined to small areas. But in these areas there was evidence of great activity, vigorous hyperplastic areas in the alveoli, with reduplication of the lining cells and even papillary formation, together with the characteristic increase of blood vessels and thinning of their walls.

To the work of Plummer we owe much that we know, of the clinical relationships of toxicity of adenoma of the thyroid, and he has shown us the effects of this type of goitre, as lasting over an average of about 14 years under ordinary conditions of life, from the beginning of the goitre, until the process is sufficiently advanced to send the patient to the diagnostitian. It seems to us certain that the unwise administration of iodine to these patients during the years when their goitres are not particularly active, has materially shortened their period of comparative safety, and that instead of coming with beginning indications of degeneration in heart and blood vessels, at about 36 years, many will show far earlier than this, advanced injury to the circulatory system ,if iodine has been extensively used. Here the drug seems to augment the case of absorption from the adenomatous areas and a quantitative poisoning easily occurs.

In our clinic the type of individual coming for the treatment of her increasing symptoms, is generally a young female, from 16 to 30 years of age, in whom a desire for a better cosmetic appearance, has led to her receiving iodine in some form. They characteristically have a goitre, rapid heart, increased blood pressure readings, some type of goitre skin, sweating hands and tremor, together with much increase of appetite and of nervousness. They have no exophthalmos and none develops. Their history reveals no evidence of symptoms of any kind referable to the goitre, until after iodine medication, over varying periods. An increasing number of these cases are seen, we believe, because certain manufacturers of iodine-containing patent medicines, have found a ready sale in the last few years for their product, in our part of the country, (which is notable for the incidence of goitre in a considerable percentage of native born individuals.)

As our interest in this type of goitre is clin-

ical and surgical rather than pathological, we state that our opinion regarding the advisability of treatment, is entirely in the direction of surgical reduction in the areas supplying the toxic products. After withdrawal of the stimulating drug, we commonly perform a double partial lobectomy, either by the method of Balfour,[9] (used for large and unwieldy growths), or in goitres of moderate size, the old Kocher operation with free drainage, leaving behind the posterior capsule and a varying amount of thyroid tissue with it, as the individual case seems to demand. We have seen no degree of toxicity in any instance, that we have felt required the preliminiary vessel ligations so important in exophthalmic goitre. Still, even with gentle, careful, rapid and well drained radical operations of this kind, occasionally a three day reaction from the procedure will ensue, in which the tachycardia, tremor and restlessness will be of a grade approaching that seen in operations during active stages of hyperplastic goitres. However, the factitious type of thyrotoxicosis has usually a sounder and less impaired constitution to aid it during the brief convalescence, and after about ten days these patients are usually ready for discharge from hospital, and many leave in a shorter period. We have operated upon fifty of these cases, within three years, and none have died.

At a later period a clearly defined group of this type of goitre, entirely of the adenomatous type, will possibly be presented, and in this an attempt will be made to adduce evidence to prove that after the free use of iodine, the period of freedom from indications of toxicity, and degeneration of other organs, is less than the classic period of their natural history, as set forth by Plummer.[9]

REFERENCES

1. Wilson: Am. Jour. Med. Sci., 1908, cxxxvi, 851-861. Jour. Am. Med. Assoc., 1914, lxii, 111-112.
2. Marine & Kimball: Arch. Int. Med. 25, 66-, June 1920.
3. McCarrison: The thyroid gland, ed. 1918, p. 40.
4. Ewing: Neoplastic diseased, ed. 1919, p. 869.
5. Crotti: Thyroid and thymus, ed. 1918, p. 280.
6. Kocher: Br. Med. Jour., 1906-1261-1266.
7. Crotti: As above, p. 282.
8. Balfour: Ann. surg., 1914, lix, 671-674.
9. Plummer: Am. Jour. Med. Sci., 1913, cxlvi, 790-796. Jour. A. M. A., 1913, lxi, 650-651. Jour. A. M. A., 1912, llx, 325-327.

DISCUSSION

DR. C. H. MAYO, Rochester, Minn.: Dr. Chapman's excellent paper very completely covers the ground with reference to "the iodin heart" mentioned by Kocher many years ago. We do not know what causes goiter. I understand it is believed something in the geologic state of certain parts of the country exhausts the iodin causes goiter, but if this were true why are so few people affected and such a large proportion of them females? The gland converts to use the iodin out of food. Some persons with myxedema cannot absorb iodin with their intestinal cells. Give them iodothyrin by mouth and they are unaffected; give it intravenously and they pick it up right away. Some types of pyogenic infection have been found in the thyroid, principally the diphtheroid bacillus.

There is a certain type of goiter, for example in girls at adolescence, for which we should give iodin in prescriptions, as demonstrated by Marine. A woman of 55 or 56 with long standing goiter has little trouble from the goiter but finally goes to a physician who puts her on iodin and the result is an iodin goiter heart. The iodin stimulates these long-standing goiters. I use the term "thyrotoxic" goiters for the cases of adenoma with hyperthyroidism.

DR. T. L. CHAPMAN, Duluth: I have nothing further to add except to thank Dr. Mayo for his discussion.

FRACTURES OF THE BASE OF THE RADIUS*

(Colles' Fractures)

ROSCOE C. WEBB, A. B., M. D.

Instructor and Associate Surgeon, University Hospital, University of Minnesota.

Minneapolis

One of the most common causes of disability which the general surgeon has to treat is the fracture of the base of the radius, generally known as Colles' fracture.

This injury was universally considered a dislocation previous to 1783, when it was first described as a fracture by Pouteau. Little attention was given to Pouteau's views, and fractures at the base of the radius were still considered dislocations in 1814 when Mr Colles accurately described the condition as a fracture, but did not gain proper credit until after his death. The Dublin surgeons seem to have been the only ones willing to accept Mr. Colles' views until about 1830, when the burden of proof seems to have shifted to the man who said

*Read before the Clinical Club of Minneapolis, November 4, 1920.

there was no fracture and Colles' views were almost universally accepted.

During the past ninety years discussion upon this subject has been largely limited to the details of causes, types, and treatment. With the advent of the X-ray a few years ago, considerable light was thrown on the subject, but a cursory glance at recent surgical literature will show that discussion upon the subject is by no means ended. The cause of this injury is generally conceded to be extreme dorsal extension due to a fall upon the outstretched and extended hand; the ligaments are more resistant than the bony tissue, which consequently gives way. Stevens,[1] in a recent extensive discussion, lays great stress upon the compressive nature of this fracture and shows that the shortening of the radius results from actual loss of substance due to compression and destruction of tissue rather than from impaction. Troell,[2] of Stockholm, in a careful study of over 200 cases concludes that they are compression fractures, or a combination of compression and extension fractures.

In a series of thirty-five personal cases collected while Assistant House Surgeon at the New York Hospital, when the late Dr. Stimson was consulting surgeon, the writer attempted to account for the variations in type which these cases present, by taking a more careful history of the injury and manner of falling; but the nature of the accident renders it nearly impossible for the patient to describe it other than as a fall upon the outstretched hand.

The usual textbook description gives the impression that these fractures present one type with slight variations and they are consequently treated alike with the usual bad results, more so than with any other fractures. Colles originally described it as a fracture which takes place about one and one-half inches above the carpal extremity of the radius. Scudder,[3] under the heading Colles' Fractures, says that a fracture of the lower end of the radius within one inch of the articular surface is common in adults. In discussing the associated complications no mention is made of the fractures involving the radio-ulnar and radio-carpal joints, although pictures are shown presenting these conditions. Jones,[4] of Liverpool, says, "In its typical form the radius breaks about three-quarters of an inch from the lower end, the lower fragment being displaced backward and usually rotated toward the ulnar side." Troell,[2] divides these fractures into nine groups based upon the anatomy. Stimson's[5] last edition places the Colles' fracture at from one-third to three-fourths of an inch proximal to the articular border and mentions the fact that it is often comminuted.

Numerous writers have called attention to the wide variations in these fractures and a distinct step in advance was made in the Bellevue[6] nomenclature, which is being widely adopted at present, in dropping the term *Colles' Fracture* and substituting for it, *fracture about wrist joint*, thus calling attention to the necessity of different and individual therapy.

Hitzrot[7] called attention some years ago to three common types into which these fractures may be divided, a classification which has proven very helpful from the standpoint of prognosis and treatment. (See Figs. 1 and 3).

A *type* follows the line of ossification between the diaphysis and epiphysis and is about 1.75 to 2.25 cm. above the radial styloid.

B type has the line of fractures entering the radio-ulnar joint.

C type is 3.75 cm. or more above the tip of the radial styloid and is found in young people.

In a series of twenty-one cases in the records at the University Hospital the following groups were found as shown in the chart. (Fig. 4).

From this chart it is seen that ten per cent of the cases are in the region described by Colles; that fifty-seven per cent lie in the region commonly described as Colles' fractures. and that thirty-three per cent lie below this

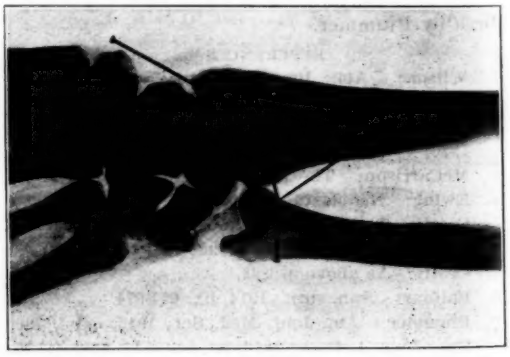


Fig.1

point. In addition, the radio-ulnar joint is involved in thirty-three per cent of the cases; the radio-carpal joint in forty-three per cent, and either one or both joints are involved in fifty-three per cent of the cases. In fifty-seven per cent the ulnar styloid is broken. It is obvious that the results can not all be the same, regardless of treatment; consequently in order to produce the best functional results the surgeon must study and interpret the x-rays and treat each case individually.

In the treatment of fractures and especially fractures at the base of the radius, the import-

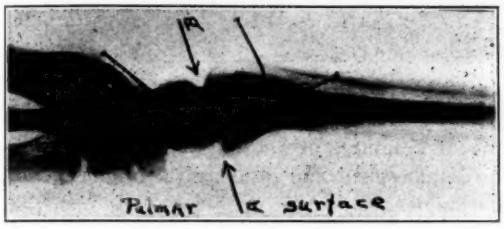


Fig. II

ance of early reduction under anesthesia can not be over-emphasized. Any one who has seen the conditions about the bone in recent fractures can readily appreciate the necessity of early reduction.

In operating upon several hundred compound fractures in evacuation hospital work in France, the writer was constantly impressed with the pathological conditions found about the ends of broken bone where the injury varied from a few hours to a few days old. Not only is there new bone formation on the second or third day, as pointed out by Macewen, but there is a pouring out of blood and exudate from the broken ends with rapid organization rendering accurate reduction difficult. There is also exudate into the surrounding muscles, fascia, tendons, ligaments synovial sheaths, and joint cavities, all of which must undergo absorption or organization. It one does not reduce early, this process renders bony landmarks obscure and if one waits for the swelling to subside, accurate reduction is seriously interfered with.

The New York Fracture Society agreed, March 7, 1917, that a fracture should be reduced within three hours, thus placing reduction of a fracture in a class with other surgical emergencies.

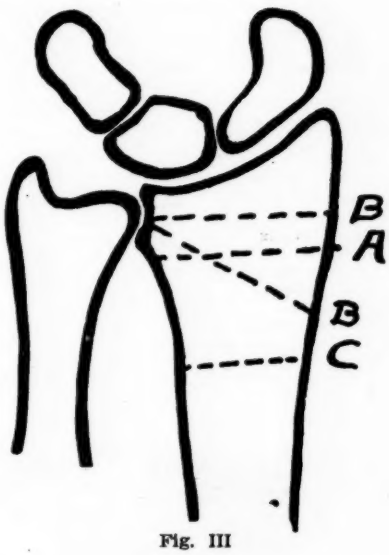


Fig. III

With anesthesia the muscle spasm as well as the pain is overcome and the surgeon is enabled to manipulate the fragments to the greatest advantage. Nitrous-oxide and oxygen is the most satisfactory anesthesia. If the anatomy of the wrist joint is recalled it will be remembered that the articular surface of the ulna is separated from the carpal bones by a triangular fibro-cartilage extending from the medial surface of the ulnar styloid to the border between the medial and distal articular surfaces of the radius. This cartilage is frequently perforated, thus permitting the escape of exudate in the B Type fracture into the radio-carpal joint. The radius, in its articulation with the os naviculare and os lunatum, forms the important part of the articulation of the forearm and the carpus.

The radial joint surface runs from behind forward and distally, (Fig. 2), so that in full extension the motion is limited by the lip of the posterior aspect of the radius. This inclination is altered in practically every fracture and the correction of the inclination of the radial joint surface is the chief failure in reduction (Fig. 5).

There are numerous methods of reduction, all of which have their place. The method which appeals to the writer in the majority of instances is that of grasping the affected extremity in the handshaking position and reproducing

Type			Ulnar broken	Styloid unbroken
A	A line (pure)	8 or 37%	5	3
	Secondary into wrist joint	3 or 15%	1	2
	Crushing	1 or 5%		1
B	Oblique line (pure)			
	Tranverse line (pure)	2 or 10%	2	
	Secondary into wrist joint	5 or 23%	4	1
C		2 or 10%		2
	Total	2112 or 57%	9 or 43%	

Fig. IV

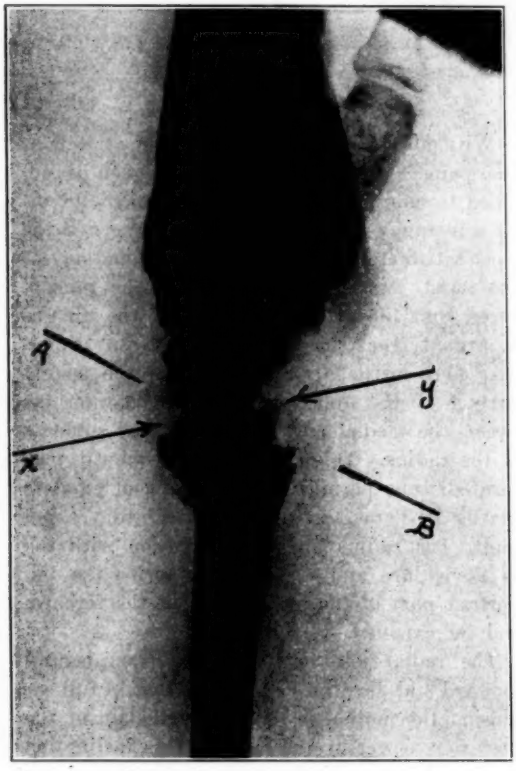


Fig. V

the deformity, with the thumb of the other hand at the lower end of the proximal fragment. When the fragments are at a right angle and the operator's thumb is in the angle, the distal fragment is brought down by a quick movement and the thumb in the angle slides down over the

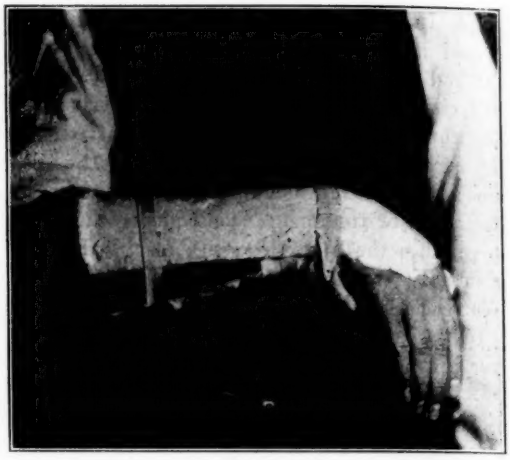


Fig. VI

line of fracture, preventing the recurrence of the displacement and insuring the alignment of the fragments.

The fracture having been reduced, the next question is one of splints and position. The molded plaster splint as devised by Dr. Stimson at the New York Hospital thirty years ago is without a doubt the most satisfactory and is far superior to any prepared splint. The position should be determined by the condition of the fracture, since no one position applies to all. In view of the fact that there is in all cases a shortening of the radius due to destruction of bone and in fifty-seven per cent of the cases a fracture of the ulnar styloid, the hand should be put up in ulnar deflection. In order to maintain the position of the radial joint surface, flexion at the wrist is a distinct advantage. Also in the flexed position the extensor tendons aid in maintaining the distal fragments in position. About three-fourths supination with the fingers absolutely free is most satisfactory. (Fig. 6 and 7). At the end of a week the hand should be brought out straight in a second set of splints, as too long maintenance in the flexed position prolongs the period of recovery.

The importance of baking and daily massage in increasing the blood supply and other healing processe in fractures is now generally recognized. The surgeon himself should give the massage during the first ten days, after which it may be done by a dependable masseur trained in fracture massage. With the plaster splints

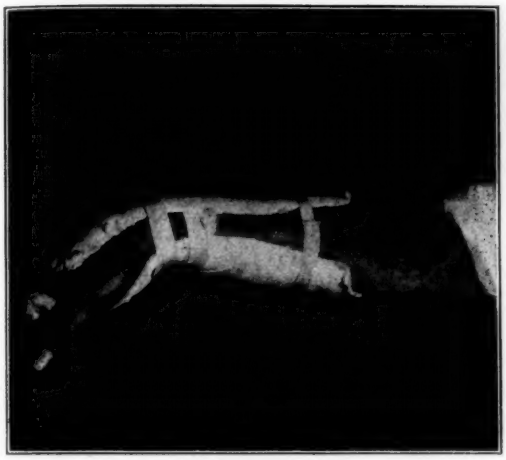


Fig. VII

the dorsal or palmar splint can be removed, and with one remaining in place massage can be given without danger. In the fracture involving the radio-ulnar and radio-carpal joints, early motion, performed by the patient under the direction of the surgeon, is essential to the preservation of the normal movements of these joints.

At the end of two weeks the palmar splints may be removed, as the fracture is well or nearly so; and in three weeks splints may be left off altogether.

CONCLUSIONS

Mr. Colles' connection with this fracture is purely of historical interest, and the substitution of the term *fracture of the base of the radius* will promote progress in study and treatment.

The fractures result from a combination of compression and extension and vary with the manner of falling and age of the patient.

These fractures should be considered as emergency cases and reduced at once under anesthesia.

The surgeon should interpret the x-ray plate himself and treat each case as indicated.

Deformity and impairment of function depend upon:

(a) The character of the line of fracture.
(b) The age of the patient.
(c) The time of reduction.
(d) The character of reduction.
(e) After treatment.

REFERENCES

1. Stevens, James H.: Compression fractures of the lower end of the radius. Annals of Surg., May, 1920, vol. lxxi, p. 594.
2. Troell, Abraham: On fractures of the forearm in the region of the wrist. Annals of Surg., Oct., 1920, vol. lxxii, p. 428.
3. Scudder, C. L.: The treatment of fractures. W. B. Saunders Co., 1915, p. 338.
4. Jones, Sir Robert: Injuries of joints. Oxford University Press, 1915, p. 107.
5. Stimson, Lewis A.: Fractures and dislocations. Lea & Febiger, 1912, p. 310.
6. The Bellevue Hospital Nomenclature: M. B. Brown Printing & Binding Company, New York, 1911, p. 112.
7. Hitzrot, James M.: Personal communication.
8. Hitzrot, James M.: Some phases of the present treatment of fractures. N. Y. State Jour. of Med., July, 1917.

MIXED TUMORS OF THE PAROTID GLAND*

By W. E. SISTRUNK, M. D.
Section on Surgery, Mayo Clinic,
Rochester, Minn.

In the treatment of mixed tumors of the parotid the best results are obtained in cases in which an early diagnosis is made and an early operation performed. In their incipiency these tumors are small, usually definitely encapsulated, and only mildly malignant. A complete removal of the growth at this stage of its development will, in practically every instance, be followed by permanent cure, and the danger of injury to the facial nerve, the worst complication to be expected, is greatly diminished when the operation is performed at this time.

Mixed tumors of the parotid constitute a very small percentage of malignant tumors of the body. They occurred in only 0.062 per cent or in one of every 1,607 patients who were examined in the Mayo Clinic from 1915 to 1919. Mixed tumors are found about eleven or twelve times as often in the parotid gland as in the submaxillary salivary glands, and they practically never occur in the sublingual salivary glands. They occur with about equal frequency on the right and left sides and in males and females.

The incipient growth is a painless small

*Presented before the Minnesota State Medical Association, St. Paul, October 1, 1920.

rounded or irregular firm tumor in the parotid which usually is discovered accidentally. If the tumor is left alone its growth varies; in some patients it is rather rapid, while in others the tumor does not seem to change in size for a number of years. Usually, however, at some time during the life of the patient it enlarges to a noticeable size. The patients in this series had noticed the growth from two weeks to forty years previous to the first operation. As long as the tumor remains encapsulated it is not highly malignant, but when its capsule is ruptured through trauma, through the growth of the tumor, or through an incomplete operation in which the entire tumor or capsule is not removed, and the cellular elements invade the tissues surrounding it, the growth usually rather quickly undergoes malignant change, and in some instances rapidly becomes a highly malignant tumor. With the rupture of the capsule metastasis is also possible and the parotid gland, with the muscles surrounding it, and the glands draining the region become invaded.

Pathologists differ with regard to the etiology of these tumors; many believe that they have their origin in fetal rests in the parotid which are encapsulated and which gradually grow. Wilson and Willis, after a study of the tumors of this nature which had been operated on in the Clinic up to 1911, believe that they are mesotheliomas. Some pathologists believe them to be carcinomas, and others, basal-cell epitheliomas. The tissue on the interior of the capsule of such tumors varies considerably; in some instances it is composed largely of epithelial elements, while in others tissue resembling cartilage seems to predominate; some tumors present a large amount of fibrous tissue. Often sections from different portions of the same tumor show very different microscopic pictures.

It is unfortunate that the parotid gland instead of the other salivary glands is so frequently involved. The condition is so slightly malignant in its incipiency that were it possible in all cases completely to excise the parotid gland without the resulting facial paralysis, as is done when such tumors are found in the submaxillary salivary glands, the percentage of cures through surgery would be about 100. On account of the possible injury to the facial nerve many patients defer having the operation performed while the growths are small and thus lose the opportunity of having it performed at the time when the chance of cure is highest and the possibility of injury to the nerve is least. Because of this danger many patients are subjected also to an incomplete operation resulting in recurrence of the growth, and possibly loss of life. Unfortunately surgery seems about the only treatment which can be expected to cure these tumors, radium and x-ray having but little effect on them. The operation usually performed is as follows:

OPERATION

An oblique skin incision is made along the creases of the neck at a point about 1.25 cm. or 1.75 cm. below the angle of the jaw and extending from the lower portion of the mastoid process for a distance of about 5 cm. or 7 cm. toward the thyroid cartilage (Fig. 1). The skin and platysma muscle are then reflected and the parotid gland exposed. Often the tumor is found superficially placed and lying along the anterior surface of the gland. In such instances a transverse incision is made in the general direction of the fibers of the facial nerve through the parotid gland down to the encapsulated tumor, which is then enucleated by blunt dissection from the tissues surrounding it. If the tumor can be removed without rupturing its capsule permanent cure results almost always. Often, however, the capsule surrounding the tumor is very thin and is ruptured in an effort to remove the growth. In cases in which this occurs it is best to seize the edges of the capsule gently with forceps and with as little tension as possible by blunt dissection separate the capsule from the surrounding tissues, making every effort completely to remove it (Fig. 2). If the capsule ruptures and collapses after a portion of its contents has escaped, a small piece of gauze may be packed into the cavity in order to distend it and define its outline, thus aiding the removal. When the capsule has been removed thoroughly the cavity in the parotid gland (Fig. 3) should be washed with salt solution in order to remove, if possible, all the cellular elements which may have escaped. It is customary with us then carefully to swab the wound with Harrington's solution (a strong

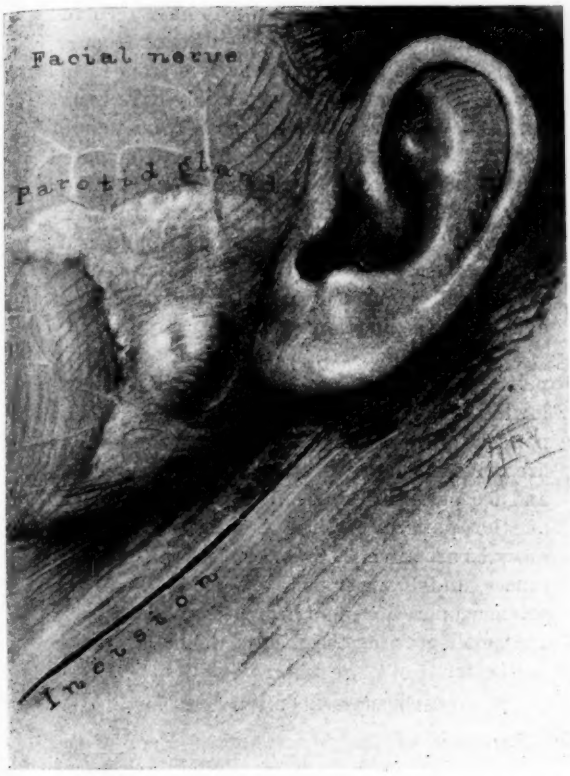


Fig. 1. Incision along crease in the neck which usually is employed in the removal of parotid tumors.

solution of bichlorid of mercury with hydrochloric acid in alcohol) in order to kill any cells that may still be lying free in the wound. The cavity in the parotid gland is packed with a small strip of gauze saturated with Harrington's solution; this pack is left in place for twenty-four or forty-eight hours.

When the superficial growth is removed by blunt dissection and no cutting is done except a small incision through the outer portion of the parotid gland in the direction of the nerve fibers, paralysis of the seventh nerve seldom occurs. In an occasional instance a temporary paralysis may occur as a result of the trauma to which the nerve is subjected during the removal of the tumor or from the pack which is left in the wound, but this usually subsides within from six weeks to a year after the operation. The paralysis which follows operation for these tumors usually occurs in cases in which the growth has attained a large size, or has ruptured and involved the surrounding tissues, or has recurred following an incomplete operation. In the latter cases a large amount of fibrous tissue which is absent in the primary operation is usually encountered at the second operation, and it is often necessary to cut tissues which, ordinarily, could be stripped by blunt dissection in the first operation. In operating on some of the recurring growths and the larger growths that are deeply placed in the substance of the gland I have, in some instances, been able to save the nerve by the following procedure: I have exposed the facial nerve by first isolating the inframandibular branch of the nerve which runs along the angle of the jaw and dissected this upward through the substance of the parotid gland to the point where the facial nerve divides into the temporal and cervical divisions. The lower pole of the parotid may then be lifted up and a finger introduced between it and the nerve (Figs. 4 and 5). By this procedure the surgeon may feel sure that the enucleation is being carried on in a plane lying above the nerve and, although a temporary paralysis usually occurs from traumatism to the nerve, it disappears within a year.

RESULTS OF OPERATIONS IN 103 CASES

I have recently reviewed the histories of 112 patients operated on in the Mayo Clinic during the years from 1915 to 1919. Five patients who had inoperable growths on admission to the Clinic and who were treated with radium, three on whom an operation was attempted but could not be completed on account of the extent of the growth, and one who was killed in France during the war were discarded from the series in studying the results obtained through operation. This left 103 patients in the series. We have had recent communications relative to all except ten; eighty-five patients (82.5 per cent) are known to be alive and eight (7.7 per cent) are dead. Six patients (5.8 per cent of 103) of the eight patients who died are known to have died from recurrence of the disease; all six patients had extensive growths at the time of operation, the glands being involved in four. Four of the six also had been operated on one or more times elsewhere. We have no data rgarding the cause of death of one patient, and in the other, death was supposed to have been

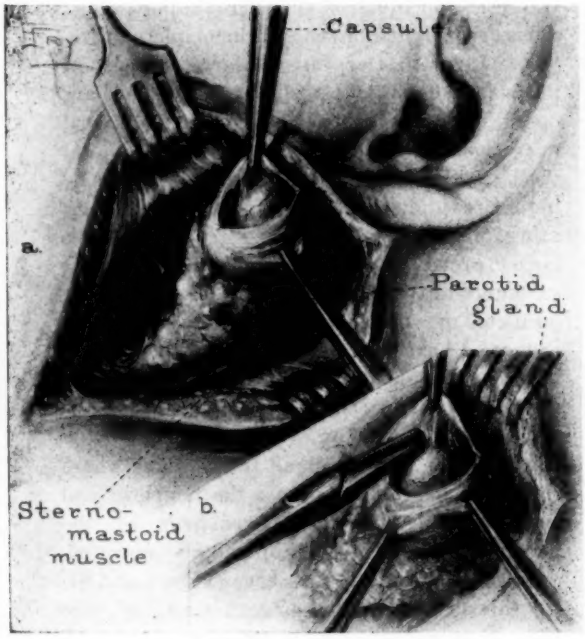


Fig. 2 a. Skin and platysma muscle reflected and the parotid tissue covering tumor incised in the direction of the fibers of the facial nerve. b. Enucleation of tumor by blunt dissection with a pointed hemostat.

caused by disease other than recurrence of the tumor.

A primary operation was performed for sixty-six (64.1 per cent) of the 103 patients. We have been able to obtain data with regard to the result in sixty of these. Fifty-six (93.3 per cent of sixty) are known to be alive and four (6.7 per cent) are dead. From forty-nine (81.7 per cent) we have recently received letters indicating that no recurrence has developed, from one to five years following operation. Eleven (18.3 per cent) are known to have had recurrences following operation; seven of these are alive with recurrences or have been operated on for recurrences, and four are known to have died; one probably died from disease other than cancer.

Thirty-seven patients were operated on for recurrences which had developed following one or more previous operations; thirty-four of these had been operated on elsewhere; three are known to be dead. Three of the patients were operated on for recurrences following one or more operations in the Clinic; one is known to be dead. The tumor is known to have recurred in fourteen. Sixteen have been heard from recently and are supposed to be free from recurrence. Three patients may possibly have recurrences. We were unable to hear from four of these patients. All of the recurrences appeared within one year after operation. However, they may appear at a much later time. In many instances the growth of recurring nodules is extremely slow. We have operated on patients with small recurring growths that had been evident for some years.

It will be noted that the results following primary operations are better than results following operations for recurring growths; 93.4 per cent of the patients with primary operations are alive, 18.3 per cent have had recurrences and 6.6 per cent are dead, while 89.2 per cent of the patients operated on for recurring growths are alive, 37.8 per cent have had recurrences, and 10.8 per cent are dead. If all the primary operations had been peformed for early and small growths the results would have been far better.

POSTOPERATIVE COMPLICATIONS

Paralysis of the facial nerve.—Four of the sixty-six patients on whom primary operations were performed presented themselves with complete paralysis of the seventh nerve which had been caused by pressure on this nerve by the tumor. In six patients the parotid gland was so extensively involved that it was necessary

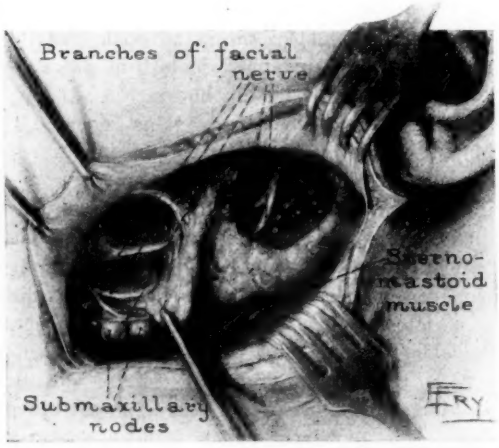


Fig. 3. Cavity in the parotid gland after removal of the tumor. Facial nerve in the bottom of the cavity.

completely to excise it, thus sacrificing the nerve. In sixteen patients a temporary complete paralysis of the nerve, which persisted for periods varying from six weeks to one year, occurred. One patient had a permanent partial paralysis and one a permanent complete paralysis. We were unable to obtain data from six of these patients as to whether paralysis had followed operation.

Four of the group of thirty-seven patients were found to have complete paralysis of the

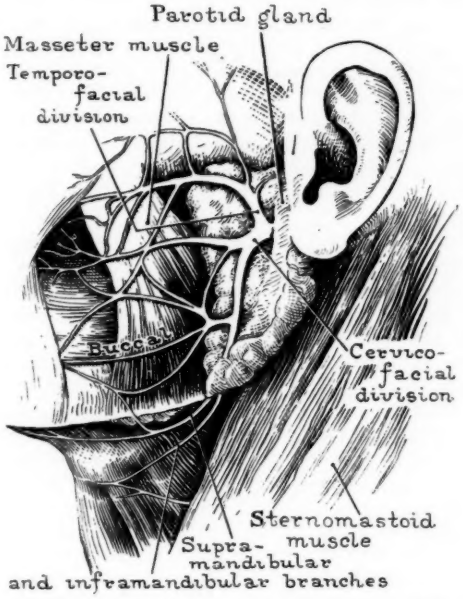


Fig. 4. Anatomy of facial nerve as it passes through the parotid gland. The anterior portion of the gland has been removed.

nerves at the time of admission; one other had a slight permanent paralysis and one gave a history of having had a complete paralysis following the operation which was done elsewhere, but this had entirely recovered. It was necessary to perform complete excision of the parotid in two of the cases in which the growth recurred; complete paralysis of the nerve ensued. Of thirteen of the patients in whom the tumor was enucleated three had a temporary complete paralysis with a return of the function of the nerve after periods varying from six weeks to one year; in two patients complete permanent paralysis followed the operation. In three patients temporary partial paralysis occurred, which persisted for periods varying from six weeks to one year, while permanent partial paralysis resulted in five. We were unable to obtain data as to whether or not paralysis had resulted in three patients.

Salivary fistulas.—Two patients had salivary fistulas after operation. One followed a primary operation and the fistula has persisted for five years and nine months; in the other patient a fistula occurred following an operation for a recurring growth and this has persisted intermittently for two years. One patient came to us with a salivary fistula which had been present for seven years and which had occurred following removal of a parotid tumor.

In closing I wish to emphasize that early diagnosis and early operation are essential in dealing with mixed tumors of the parotid in order to minimize the danger of injury to the seventh nerve and to diminish the mortality.

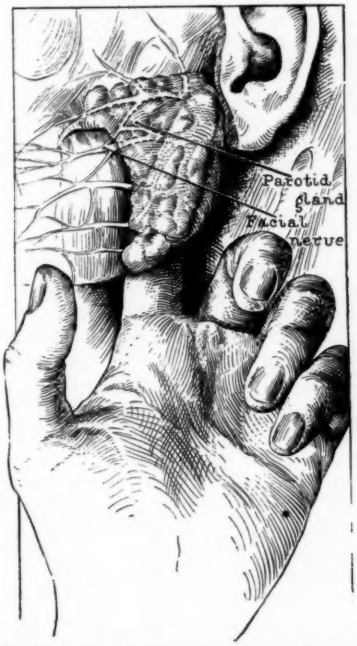


Fig. 5. Method sometimes used in trying to prevent injury to the facial nerve. The facial nerve has been exposed and separated from the anterior portion of the parotid gland. A finger is kept in front of the nerve while the tumor is being removed.

DISCUSSION

DR. MOSES BARRON, Minneapolis: Dr. Sistrunk's paper presents some very interesting features some of which should be emphasized. Tumors of

the parotid are a rather peculiar type of neoplasm not to be found in other parts of the body except in the other salivary glands.

The importance of operating early on mixed tumors of the parotid is just as essential as that of operating on cancer in different parts of the body. The earlier the operation the better the results obtained.

Dr. Sistrunk emphasized the fact that these tumors become malignant. In the early stages they are not very malignant. As long as they remain encapsulated they do not metastasize. It has been found that in the early stages when the capsule is still unruptured, the tendency for these tumors is to remain localized, and to behave very much like basal-cell carcinomata. Therefore, any surgical procedure in which the entire tumor is not removed is worse than not attempting surgery at all, since the portion which remains will very shortly spread into the surrounding tissues and into the lymph nodes draining the part. Consequently, in operating on these tumors it is very important that the removal be complete. Tumors with unruptured capsules seldom metastasize, which fact pleads for an early removal.

These tumors occur at all ages; they have been described in infants nine months old, and they occur at the age of 75 years or more. They are most common between the ages of 20 and 40.

Some of the tumors develop rapidly in the course of a few years and become very malignant. Others develop more slowly and persist for a considerable period of time.

The character of this tumor is quite different from tumors in other parts of the body probably because of the glandular secretion. It is a question whether this type of tumor is a true mixed tumor. Although we find structres resembling epithelium, mucoid tissue, connective tissue and cartilage, the origin of these elements is under debate. A number of investigators classified these tumors as endotheliomata arising from the endothelial spaces within the gland, but later researches of men like Krompecher, Eheric and Ewing have demonstrated fairly well that these tumors are epethelial in origin. They arise either from the epithelium of the acini and ducts of the gland or from misplaced embryonal rests and branchial remnants. The origin of the mucous tissue as well as the cartilage has been traced directly to the epithelial cells. Occasionally portions of the tumor present areas of squamous epithelium with prickle cells. The presence of prickle cells in some of the tumors suggests strongly that these tumors arise from epithelium rather than from endothelium.

The epithelial cells often arrange themselves in gland-like structres. These have been traced out and have been found to show an anatomical relationship to the ducts and acini of the gland.

The appearance of cartilage and mucoid tissue in these tumors has lead pathologists to classify them among the mixed tumors. But careful studies have shown that it is the epithelial strands themselves that gradually become transformed into the mucoid and cartilagenous tissue. Such transformation is rather unique and is apparently not found in any other part of the body.

DR. A. R. COLVIN, St. Paul: The interest in parotid tumors outside of the operative technic lies largely in the question of pathology. There are perhaps no other tumors which prove to us how little we really know about tumors. We find a cellular structure, epithelial or endothelial, in the parotid gland, we shell that tumor out like a pea out of a pod and inside of a year or two we find a recurrence. We have as a matter of convenience divided parotid tumors into mixed tumors, enchondroma, lipoma, sarcoma, but histologically we cannot be sure of their behavior. I know as a matter of personal experience that we are never safe with parotid tumors. I know from my own personal experience that recurrences are seen even among the most encapsulated tumors within two or three years. There is no question about the fact that the earlier such tumors are removed the less liable, of course, we are to have recurrences.

DR. H. B. SWEETSER, Minneapolis: I recently had a case of mixed tumor involving the labial glands. The patient presented himself with a tumor of the right upper lip projecting beyond the tip of the nose. He also had two other small tumors on the other side of the upper lip. A diagnosis of fibroma was made, but histologic examination of the larger tumor showed it to consist of epithelial cords, cartilege, bone, fibrous tissue, and, what the pathologist said was a very rare association,—pearls of squamous celled epithelioma. The real diagnosis, therefore, was a mixed tumor of the labial glands. In a recent article on mixed tumors of the parotid gland, the author described a mixed tumor of the labial gland, very similar to mine. It is well, therefore, to remember that mixed tumors are found elsewhere than in the salivary glands, notably in the labial glands. With this fact in mind, one may possibly avoid an unnecessary mutilating operation.

DR. W. E. SISTRUNK, Rochester (closing): I wish to thank the gentlemen who have discussed the paper. I was interested in their remarks regarding the pathology. I did not touch fully upon this because I know that considerable difference of opinion exists among pathologists as to the etiology of these tumors.

I think Doctor Sweetser is correct in believing that these tumors occur in other regions. Doctor New recently reported a series of these tumors which had occurred in other regions. We have felt that when an encapsulated tumor of this type is completely removed the chances for recurrence are extremely small. Most of the patients in whom we have seen recurrences were those in whom the tumor was ruptured during removal. I believe if any portion of the capsule is left, a recurrence is likely to occur.

POLYCYTHEMIA VERA*

By
ERNEST T. F. RICHARDS, M. D. and
EDGAR T. HERRMANN, M. D.
St. Paul, Minn.

A variety of conditions may produce an erythrocytosis or increase in the red blood corpuscles above the normal. When dependent upon a discoverable cause such a red cell increase is grouped under the secondary polycythemias, which may be classified as follows:

(a) Relative increase of red corpuscles due to plasma diminution caused by deficient fluid intake or marked fluid loss, as in the case of severe diarrhea, diaphoresis, diuresis, etc.

(b) A local increase of red blood cells due to asphyxia of the tissues.

(c) Compensatory increase in the red cells due to pathological processes, as in cardiac or renal diseases, respiratory diseases, portal or other venous thromboses, chemical poisons such as acetanilid or phosphorous, and finally, methemoglobinemia.

(d) Physiological increase in the red corpuscles following violent exercise or living in high altitudes.

In addition, however, to those conditions in which the rise in red corpuscles is secondary, we occasionally meet with an excessive and persistent increase of erythrocytes for which there is no discoverabe cause. To this small but important group of unexplained erythrocytoses the term polycythemia vera, or erythremia, is applied. It is this primary type to which the French investigator Vaquez first drew our attention in 1892, and which Osler in 1903 established in his well known article as a distinct clinical entity. Osler associated with the polycythemia in this primary group certain other clinical findings which have become classical, namely, a chronic peculiar type of cyanosis, a moderate enlargement of the spleen, weakness, headache, and vertigo.

Polycythemia vera may be defined as a disease in which the mechanism determining a normal red cell count has been damaged, the normal balance between red cell production and destruction having been overthrown, resulting in an abnormal erythrocytosis with its attending phenomena.

Three types of primary polycythemia are at present recognized:

(a) The type described by Vaquez and Osler showing a normal blood pressure and an enlarged spleen.

(b) A second type with normal blood pressure values but no palpable spleen.

(c) The type described by Gaisbok, lacking a palpable spleen but showing a definite hypertension.

Moderate kidney disturbances, especially the finding of albumen and casts in the urine, may occur in all three types.

PATHOGENESIS

Concerning the pathogenesis of polycythemia, theories may be divided into several groups the most important of which are:

I. That the disease may be due to a primary irritation of the bone marrow. In favor of this is the evidence of bone marrow activity described in many cases, i. e., the occurrence of nucleated red cells in the blood stream, an absolute increase in the leukocyte count, findings of myelocytes, as well as autopsy findings of hyperplastic bone marrow. Osler, Zeller, Falta, Tancre and Bottner support this view. Pathologically six of the cases collected by Osler showed intense hyperplasia of the bone marrow, "myelomatosis rubra," to employ his phrase. He also found nucleated red cells were present. Falta reports leukocytosis, the findings of myelocytes and myeloblasts with a basophilia of 6 to 8 per cent. Bottner supports this view because of marked therapeutic success obtained by X-ray therapy of the long bones.

II. A theory that the disease may be due primarily to a lack of normal erythrocyte destruction, placing the cause variously in the liver, spleen or in the blood itself (Pribam, Lowy, Herrenheiser). Data to support this view will be discussed in connection with the present case.

III. In addition to the two chief groups just mentioned, it may be added that deficient quality of hemoglobin, disturbed balance of blood and tissue gas exchange, and certain vague vasomotor changes, have also been considered as possible etiological factors.

The following case is presented especially be-

*Read before the Minnesota State Medical Association, St. Paul, October, 1920.

cause of its important bearing on the pathogenesis of polycythemia vera:

Male, age 54.

Family history: Negative.

tal and optic. Frequent attacks of vertigo.

Family history: Negative.

Occupational history: Pattern maker.

Past history: Pertussis. Chronic pharyngitis. Slight deafness.

Cardio-Respiratory: Negative.

Gastro-Intestinal: Appetite good. For the last four years has had occasional distress immediately or one-half hour after meals. Bowels regular.

Genito-Urinary: Negative.

Neuro-Muscular: Negative.

Marital: Married 27 years. 3 children. Denies venereal infection.

Habits: Negative.

Weight: Present, 150; no recent loss.

Present illness: In May, 1913, both the patient and his family noticed that the color of his face had changed from a normal tint to a cherry red. The patient thinks that this change of color took place in a relatively short time. In the following month he had a sudden severe attack of vertigo and had to be assisted into bed. While in bed he vomited and this was followed by a terrific headache, chiefly frontal and optic. It felt to the patient as if knives were cutting into his eyes and at the same time the sense of pressure was so great that he felt as if his eyes might burst from their sockets. A doctor was called who prescribed some capsules with temporary relief.

From this time on the headaches came with increasing frequency and violence until finally the patient dreaded going to bed at night because of them. They were usually optic and hemicranial, the right side being chiefly involved. Lying down made the pain insupportable. Drinking black coffee and standing erect were the only measures that gave any relief. During this period the purplish-red color of the hands and face became extreme.

The patient consulted us in January, 1920, complaining of almost incessant, violent headache and frequent attacks of vertigo. He stated that the bluish-red cyanosis then present was made worse by exercise and hot weather.

Physical Examination: January 17, 1920. A well nourished individual presenting a most striking bluish-red cyanosis of face, ears, hands, and to some extent, feet.

Eyes: Pupils react slowly but fully to light and accommodation. Conjunctivae are suffused.

Ophthalmoscopic examination: Fundi deep red, marked filling of veins, discs normal.

Mouth, tongue and pharynx show an extreme purplish-red color.

Heart and lungs: Negative.

Blood pressure: Systolic 144, diastolic 86.

Abdomen: Spleen just palpable at costal margin.

Extremities: Negative except for bluish-red color.

Deep reflexes: Hyperactive but equal.

Fluoroscopic examination of heart and chest negative.

LABORATORY FINDINGS

Blood Examinations

Date		Red Cells	Leucocytes	Hemoglobin
1-17-20	.....	7,840,000	6,800	115% (Dare)
1-29-20	(a) before exercise.....	9,480,000	.....	.....
	(b) after exercise.....	9,696,000	.....	.....
2- 2-20	550 c.c. blood removed			
6 hours	after venepuncture:			
	(a) before exercise.....	7,040,000	.....	.....
	(b) after exercise.....	7,496,000	.....	.....
2-21-20	.....	7,536,000	.....	115%
3-16-20	.....	7,736,000	.....	105%
3-29-20	.....	6,888,000	7,000	108%
4-12-20	.....	.....	11,000	.....
4- 5-20	250 c.c. blood removed			
4-19-20	.....	7,144,000	.....	.....
4-30-20	.....	.....	.....	107%
5-24-20	200 c.c. blood removed	6,782,000	.....	102%
6-15-20	250 c.c. blood removed			
7-17-20	200 c.c. blood removed			
7-29-20	150 c.c. blood removed			
8-21-20	.....	6,784,000	12,800	96%

Smears: Red cells normal throughout; no nucleated red cells, Howell-Jolly bodies or myelocytes.

Differential of leucocytes: P.M.N. 63%, Lym. 28%, Trans. 4%, Mono 2%, Eos. 1%, Bas. 1%.

Coagulation time:

3-29-20..... 3 min. (normal 3-5 min.)
4-12-20..... 4¼ min.
4-19-20..... 5½ min.
4-30-20..... 4¼ min.

Bleeding time:

2 min. (normal 2-5 min.)

Blood Chemistry

Total Solids: 24.7% (normal 20%).
Urea nitrogen: 15.45 mg. per 100 c.c. (normal 12-15 mg.)
Creatinin: 2.85 mg. per 100 c.c. (normal 1-2.5 mg.)
Blood sugar: 0.10% (normal 0.08-0.12%).
Total nitrogen of blood: 1800 mg. per 100 c.c. (normal 3000 mg.)
Non protein nitrogen: 34.8 mg. per 100 c.c. (normal 25-30 mg.)
Sodium chloride: 0.57% (normal 0.65%).
Cholesterol of blood: 0.312% (normal 0.15%).
Cholesterol of serum: 0.268% (normal 0.15%).

Urine: Shows a trace of albumen, an occasional hyaline and granular cast, a few red cells and leukocytes.

Phenolsulphonethalein: Excretion normal.

SUBSEQUENT COURSE

During February and March the patient was kept quiet, for the most part in bed. Following the removal of 550 c. c. of blood by vene-

puncture on February 2nd there was a marked subjective improvement. The patient stated that before withdrawal of the blood he had dreaded lying down because of the intensity of the headache, but that a few hours after the venepuncture he felt completely relieved not only from the headache but also from the sensation of fullness in the head and the vertigo. A distinct effect on the red cell count was noted after this venepuncture, the cells dropping (as shown under "Laboratory findings") from 9,480,000 before venepuncture to 7,040,000 six hours after the venepuncture.

Up to date, that is, during a period of eight months, the marked subjective improvement has continued. A number of venepunctures have been performed during this time. The patient has been able to fully carry on his work now for several months without any return of the headaches or of the vertigo.

DISCUSSION

This case fits best into type B of the primary polycythemias, the spleen when the patient was first seen being only just palpable and later not discernible to touch. The cyanosis and subjective symptoms are of course typical, while the red count and hemoglobin values are of average height for cases of erythremia. The highest values as yet reported are those given by Tancre whose red count reached 14,200,000, with a hemoglobin (Sahli) of 178 per cent and a white cell count of 18,000. A point of interest is Tancre's findings of abundant normoblasts. In the present case there is no absolute increase of leukocytes, nor is there evidence of erythropoietic activity, facts in accord with the observations of Herrenheiser, Fraser, Pribam and others.

Tancre reports a lengthened bleeding time with a coagulation time in the upper limits of normal. Our results are within the normal limits for both.

Total solids of the blood in our case are above normal, the percentage being somewhat higher than that reported by Herrenheiser. Total nitrogen is low. Von Jaksch and Lowy report an increased nitrogen content of the whole blood with a low nitrogen content of the red cells, the latter finding, according to these investigators, indicating old undestroyed red cells "physiologically under par."

In the present case exceptionally interesting values were obtained for blood and serum cholesterol, both being increased in amount to almost twice the normal. Rothchild and Falsen believe the liver to be the regulator of cholesterol-metabolism, the cholesterol being kept at a more or less constant level by its excretion through the bile. The action of cholesterol is strongly antihemolytic, and significantly Gorham and Myers report low cholesterol values in the blood plasma of patients having pernicious anemia. Pribam, who also found high values for serum cholesterol, believes that in erythremia the destruction of red corpuscles in the spleen is interfered with because of the increased serum cholesterol. He showed such serum to possess greater resistance to hemolytic poisons than normal serum.

Lowy corroborates Pribam's findings. He believes that splenectomy in erythremia either makes the condition worse or does not affect it, and he assigns a secondary role to the spleen as a possible causative agent. His conclusion is that increased serum cholesterol prevents red corpuscle destruction and that the piling up of old red cells in the blood stream finally acts as a stimulus to the blood-forming marrow with a consequent appearance in the blood of immature red cells. These results, together with the findings in pernicious anemia, when compared with the figures obtained in the present case both as to blood and serum cholesterol, point strongly toward insufficient red corpuscle destruction as a causative agent of at least one type of polycythemia of which the present case is a striking example.

Considering all available data, therefore, it seems quite possible that polycythemia vera may be produced by two distinctly opposite processes:

I. By pathological activity of the erythropoietic system with abnormal production of red cells due to a stimulus of unknown origin and doubtful point of attack.

II. By lack of normal red cell destruction, which in a certain number of cases, of which the present is an example, may be induced by an abnormally high serum cholesterol content, the latter perhaps indicating a functional disturbance of the liver.

Therapeutically the above division is likewise justified. In the present case venepuncture was resorted to with excellent results. The severe headache, dizziness, subjective sensations of ting-

ling, numbness, etc., which the patient complained of when first seen, disappeared almost entirely after the first removal of 500 c. c. of blood. Altogether nine venepunctures were performed, the last eight chiefly in the hope of influencing the red count since the subjective symptoms had not reappeared. After the first venepuncture it will be seen that the red cell count dropped by two million while the hemoglobin fell from 115 per cent to 96 per cent. Cases in which there is definite evidence of bone marrow activity are often not relieved or, as in Tancre's case, are even made worse by venesection.

TREATMENT

The treatment of polycythemia in general is symptomatic at best, the measures employed being as follows:

(a) Venesection. Giving in the present case excellent results.

(b) X-ray. Bottner sums this up well by saying: 1. That the spleen should receive stimulating doses of rays to promote erythrolysis; 2. That the long bones should receive big doses of rays to paralyze erythropoietic production.

X-ray treatment of the spleen alone has yielded disappointing results, that of the long bones, on the other hand, good ones.

(c) Radium. Falta reports very favorably on the use of radium particularly when applied over the spleen and ribs.

(d) Drugs. In a recent communication Eppinger and Kloss lay great stress on the use of phenylhydrazin and toluylendiamin. They gave phenylhydrazin in doses of 2 to 10 c. c. of a 1 to 5 per cent solution subcutaneously with great improvement subjectively and a marked drop (to normal) of the red count. These two drugs aid in the destruction of red cells. Benzol, on the other hand, checks production.

CONCLUSIONS

1. A strict differentiation between the primary and secondary polycythemias is of great importance.

2. In cases exhibiting severe headaches or vertigo, polycythemia as a possible cause should be more frequently considered.

3. In the present case of polycythemia vera we are dealing with diminished red cell destruction which is apparently dependent upon an increased cholesterol content of the blood serum.

4. The importance of determining bone marrow activity on the one hand, or diminished erythrocyte destruction on the other, is essential in every case of polycythemia as a practical basis for therapy.

BIBLIOGRAPHY

Friedenwald—Contrib. Med. & Biol. Research, Sir W. Osler, N. Y., 1919, pp. 495-501.
Bottner, A.—Dtsch. Med. Wchnschr. Leipzig, Berl., 1920, XLVI, p. 66.
Falta, W.—Wien Klin. Wchnschr., 1919, XXXII, p. 157.
Herrenheiser—Dtsch. Archiv. f. Klin. Med. 130, V. 1919, pp. 315-330.
Freund, H.—Munch. Med. Wchnschr., 1919, LXVI, p. 84.
Gatrein & Singer—Zentralblatt f. Inn. Med. Leipzig, 1918, XXXIX, pp. 423-435.
Tancre—Dtsch. Archiv. f. Klin. Med. Leipzig, 1917, CXXIII, pp. 435-452.
Zadek—Berl. Klin. Wchnschr., 1918, LV, p. 1193.
Zeller—Ibid. pp. 1218-1220.
Eppinger—Therap. Monatschr. Berl., 1918, XXXII, pp. 322-326.
Ludin—Ztschrf. f. Klin. Med. Berl., 1917, LXXXIV, pp. 460-476.
Schneider—Berl. Klin. Wchnschr., 1918, LV, p. 415.
Arnsperger—Munch. Med. Wchnschr., 1917, LXIV, p. 814.
Curschmann—Med. Klin. Berl., 1917, VIII, pp. 35-38.
Von Kogerer—Wien. Kl. Wchnschr, 1917, XXX.
Rosenfeld—Berl. Klin. Wchnschr., 1917, LIV. p. 1050.
Schmidt—Med. Klin. Berl., 1918, XIV, p. 553.
Pribam—Festschr. f. R. v. Jaksch, Prag., 1912.
Pappenheim—Grundriss d. Hem. Diag. v. Blut Untersuch. Leipzig, 1911.
Rexford—Arch. Int. Med., 1916, Vol. XVII, pp. 415-419.
Lowy—Med. Klin., 1912, 36, p. 1464.
Osler—Am. Jour. Med. Sc., Vol. 126, p. 187.
Osler—Lancet, Jan. 18, 1908.
Fraser—Lancet, Vol. CXCVI, No. 4983, March 1, 1919.
Lamson—Jour. Pharm. & Exp. Ther., Vol. 9, pp. 129-154.
Hawk—Prac. Physiol. Chem., Philadelphia, Blakiston, 1918.
Marsh—Med. Clin. North America, 1919, III, 741.
Gradwohl—Blood, Urine, Chemistry, St. Louis, Mosby, 1920.
Gorham and Myers—Arch. Int. Med., 1917, No. 4, p. 599.
Rothschild and Felsen—Arch. Int. Med., Vol. XXIV, No. 5, p. 520.

DISCUSSION

DR. H. Z. GIFFIN, Rochester, Minn.: Dr. Richards' observations concerning the cholesterol content of the blood are important, and his contrast between pernicious anemia and primary polycythemia is very interesting. In that connection I might say that I have

used polycythemics twice in transfusing patients with pernicious anemia and no harm has resulted, nor was there especial benefit. This paper is of importance to all of us because of the fact that cases of polycythemia frequently are not recognized. Many mild cases of polycythemia have been diagnosed as neurasthenia on account of their nervous symptoms, and some typical cases have not been recognized at all. A few have been diagnosed brain tumor on account of headache and the fact that they had developed choked disc. Patients with polycythemia are apt to have cerebral hemorrhage. The importance of the nervous symptoms, the importance of the recognition of the type of erythrosis (the color is not due to a true cyanosis), and the importance of the eye signs are definitely established.

Venesection repeated at intervals gives symptomatic relief and this is all one can hope for at present in the treatment of the condition. Splenectomy has not been done in any of our cases. Four instances have been reported in the literature, but the results are not conclusive either for or against splenectomy.

DR. H. L. ULRICH, Minneapolis: In the discussion of this disease the most fundamental point is that we have an increase in the reds; the enlarged spleen, the increased blood pressure, the absence of splenomegaly, or again the absence of blood pressure and no splenic enlargement are but variants around this central fact. When one considers the findings with the increased density of the body fluids a good many of the symptoms could be explained on the basis of a true plethora expressing itself in this or that organ. For instance the eye symptoms, papilledema going on to choked disc, or again the severe headaches are most likely due to pressure i. e. plethora. In going over the literature the variety of group symptons are rather confusing. We have cases where vasomotor disturbances go with severe hemorrhages, or thrombosis; and as Dr. Giffin mentioned there are cases in which there is a definite endocrine complex. It is this variety of sympton complex which makes the classification very confusing and for this reason it is well to consider a fundamental increase of the reds without any explainable cause, such as disease of the lungs, or from chemicals, a form of polycythemia vera. The liver and bone marrow have been mentioned in the etiology of this disease. The spleen also has come in for its share as a causative agent. The spleen is supposed to send out a hemolytic substance which stimulates the bone marrow to overproduction. This theory is based on the same principle as that of overproduction of antibodies following the stimulation of a toxin. In this connection we must not forget the recent contribution of Lamson, who has been able to produce temporary polycythemia in animals and man by the injection of adrenalin. Then there is another point; we have an increase in volume of carbon dioxide and oxygen content of the blood. This volume increase may have some effect on endocrine secretion. This is however mere speculation. Dr. Richards' idea that the increased cholesterol content of the blood due to impaired liver function may inhibit red cell destruction is a novel and a distinct contribution to the etiology.

In the matter of treatment some have adopted splenectomy, some have used radiation of bones, and some have used bleeding. These points have been dwelled upon by Dr. Richards. I think if we determine the type based on symptons and the possible etiological factor, it will determine our approach to treatment.

DR. L. G. ROWNTREE, Rochester: I am greatly interested in this paper, and particularly in Dr. Ulrich's remarks in relation to Lamson's work on polycythemia. This work was begun in our laboratory at Johns Hopkins. Dr. Lamson attempted to mark the red cells in such a way that he could identify them again after injecting them into other individuals in an attempt to determine blood volume. Simultaneously we tried out other lines of determining blood volume. The blood volume should be remembered in considering polycythemia. We have two factors, the cells and the plasma. We usually think of polycythemia as being due to increased blood cells, without distinguishing whether or not this is relative or absolute. In the blood counts we have to consider the blood plasma as well as the cells. In our blood volume method we determine first the plasma in the body, and from the relation of plasma and cells as shown by the haematocrit we arrive at the total volume. By determining blood volume it is impossible to differentiate in a given case whether the polycythemia is due to a decrease in the plasma body resulting in a relative increase in the blood cells, or whether it is an absolute and relative increase of the red blood cells. This is a fundamental conception and should be taken into consideration in any theory which has to do with the etiology of polycythemias. I might say that polycythemias of both types have been described.

Dr. Schneider's duodenal content methods might be used to great advantage in determining whether red cells undergo less destruction in polycythemia.

I should like to speak of a case of polycythemia I saw last week with Dr. Giffin. The patient two years ago showed a marked polycythemia. He now returns with a normal blood picture, but he has developed true gout, with tophi formation. For the gout we are using radium, with remarkable results. Dr. Richards mentioned radium as a treatment for polycythemia. We should differentiate the way it is used. Radiation over the spleen might decrease the red cell count, but if we use radium as a sipping cure, as it is usually used in gout, an increase in the red blood count will probably follow.

DR. RUSSELL M. WILDER, Rochester: I should like to ask Dr. Richards whether in the course of his studies he has observed an increase in the sugar content in any of his cases. I know in this particular case none was found, but in this connection it may be interesting to mention a case at the Mayo Clinic which will be reported in more detail, a case of poly-

cythemia vera and true diabetes mellitus occurring simultaneously. This is the first instance to my knowledge, after looking through the literature fairly carefully, of the association of these two diseases. Leukemia has been noted in diabetes, but not polycythemia vera.

DR. GEORGE DOUGLAS HEAD, Minneapolis: A case that I have now under observation is that of a physician's wife who apparently has true polycythemia, in which the red count is about eight million and the haemoglobin about one hundred and twenty-five per cent. In this case, the most careful examination which we could make disclosed no etiological factor except that the patient gave all the tests for tuberculosis, including the subcutaneous test. It is a point to bear in mind that primary tuberculosis of the spleen may simulate very closely polycythemia and give a typical picture of this disease. This patient was given radium over a period of a month, the spleen being exposed in various parts, with marked diminution in the number of red cells and haemoglobin. After observing the patient for a year's time and noting the remarkable improvement with the use of radium, I was inclined to report this case as a cure of a case of polycythemia by the use of radium. However, the rules of conservative observation led me to wait a while longer, and last winter the patient returned with the red count at about the same figure as when I had first seen her. It is quite possible that with polycythemia we have a disease which runs exacerbations and remissions, much as does pernicious anaemia. The careful chemical studies which Dr. Richards has made upon the blood in connection with his interesting case is a splendid object lesson to each one of us and clearly indicates that not all of the resources of investigation have been exhausted by any means in the study of these obscure blood conditions.

DR. ERNEST T. F. RICHARDS, St. Paul (closing the discussion): I appreciate very much the interesting discussion this paper has elicited. The case is reported not because of its clinical interest, as the subject has been well covered clinically in previous articles, but because we especially wish to bring out the important bearing of the serum cholesterol content on red cell determinations and the inhibiting effect of a raised serum cholesterol upon the amount of erythrocyte destruction. We feel this case especially emphasizes this important phase in the pathogenesis of polycythemia.

USES AND ADVANTAGES OF ELECTRICALLY DRIVEN BONE SURGERY EQUIPMENT IN HEAD SURGERY, WITH PARTICULAR REFERENCE TO EAR, NOSE AND THROAT OPERATIONS*

L. W. MORSMAN, M. D.
Hibbing, Minn.

That more oto-laryngologists of this section may avail themselves, and give their patients the benefit, of the electrically driven instruments, which will be briefly described and their uses given, it seems opportune to discuss this subject today.

The advantages of this method over other methods, as, chisels, gouges, curets and punches, are so great and varied that it seems surprising to the author that any head surgeon should today be without such equipment.

One of the greatest advantages of such technic is the amount of time saved, allowing the operator to do operations which are confined mostly to bone with the saving of time, varying from twenty to sixty per cent, depending upon the type of operation done. This allows for a shorter anesthetic, the advantages of which you are all well aware. The use of the burr has a hemostatic action, so that the operator does not have to wait for sponging. This also saves time. The dexterity with which one is enabled to work about in dangerous areas, especially in the mastoid operation, is a great time saver. But the greatest factor in time saving is the rapidity with which bone may be taken down with the newer instruments. The most important advantage of all is the greater protection and safety for the patient. It has been repeatedly demonstrated that bone may be taken down completely and rapidly against the dura, lateral sinuses, seventh nerves, jugular bulb, or like important structures, and that soft tissue, if at all movable, will be actually pushed aside by the burr without injury. A simple test, that will inspire confidence in the new operator, is to pass the rotating burr over the extended gloved hand and note the rubber glove is uninjured. The bony walls of the cochlea and semi-circular canals may be actually taken down or removed,

*Read before the Minnesota State Medical Association, October, 1920, St. Paul, Minn.

without entering the lumen of these structures, as has been demonstrated on the cadaver, as well as the entire dissection of the seventh nerve, without injury to its sheath.[1]

A most important factor is the elimination of surgical shock—this partly by reduced operating time, partly by reduced hemorrhage and largely by elimination of the continued hammering blows and their effect upon the central nervous system in removing bone with chisels and gouges. In doing skull trephine for decompression, or other cranial operations, time and shock may be saved the patient, and much labor the operator, by using the skull trephine and guard attachment, followed by the spiral osteo tome.

There are really no disadvantages or objections, if a good equipment is used. Much has been said about burning the bone from the heat of the burr. The burr does not become hot if a number are used and rapidly changed. This change is of a distinct advantage, as the surgical nurse can keep the grooves thus readily cleaned. Beck has answered this objection in detail from his studies in the laboratory of the North Chicago Hospital. At different stages of mastoid operations, specimens were taken for microscopic study, and he reports that such objection is based on myth.

As to the use of equipment, there are many engines and attachments being offered to the profession by the instrument makers, and many of these, no doubt, will prove very satisfactory instruments. Some of these types are best suited for some particular operation. The one used by the author, and found most advantageous for all types of work, is the Halle-Mueller universal bone surgery equipment. This equipment is particularly adapted to clinics and hospitals and can be used with equal benefit by the general surgeon, the orthopedist, and the oto-laryngologist. The original was designed by Dr. Max Halle of Berlin and has a handpiece that is far superior to other models. The chief factor of the handpiece lies in the fact that the instrument is in operation only while the index, or trigger, finger is held back. It has a natural hold, or pistol-grip, and there are no projections from above to interrupt the vision. This is particularly desirable when working with the use of the head-mirror and reflected light. All attachments may be boiled without injury, with the other instruments.

A perusal of the literature finds the origin of the use of such equipment in doubt, but that it was first adopted from the trades by the general surgeon for reducing rough edges of bone is apparent. It was adopted secondly by the dental surgeon and thirdly by the oto-laryngologist. Credit is given to Jansen for first using the electrically driven burr in mastoid surgery and to Allport for taking it up first in this country and extending its use to other operations about the head. Members of this section, who are doing general or orthopedic surgery, may be referred to the articles of Albee,[2] Magnuson, Hartley, Kenyon, Creyer and Davidson. Oto-laryngologists, who most enthusiastically advocate electro-motor instrumentation, are Halle, Jansen, Boettcher, Allport, Beck, Robinson and many other well known men. In the literature produced by this group are detailed twenty-three operations, as practised by them, in which they claim great superiority for this technic. To attempt to go into all these operations would take entirely too much time for the brevity of this paper. We will therefore touch upon some of the vital points of two or three, especially adaptable to electro-motor instrumentation on account of the bony structures in which the operations are performed. The same principles may be applied to all.

One of the most satisfactory operations to perform is the radical antrum, or modified Caldwell-Luc, operation. As performed by the author, ether anesthesia is used, transferring to the use of the Beck-Muelle ether vapor apparatus. Incision is made at the gingio-labial margain, immediately over the base of the infected antrum, a periosteal elevator quickly exposing the bone. A No. C round burr opens the antrum directly from in front and several rotations of the rapidly moving burr will take off the necessary amount of wall to work through. The mucous membrane is then incized and the cavity sponged and such diseased tissue as found quickly removed with the curet,

[1]Beck, Joseph C. A plea for the electrically driven burr in bone surgery of the head. Trans. American Academy of Ophthalmology and Oto-Laryngology, 1914.

[2]Albee, Fred H. Bone Graft Surgery, W. B. Saunders Co., 1915. Orthopedic and Reconstruction Surgery, W. B. Saunders Co., 1919.

being careful to remove no mucosa which is not diseased beyond prompt regeneration. The mucous membrane, including periosteum, is removed from that portion of the nasal wall lying beneath the attachment of the inferior turbinate body. One essayist,[3] at the last meeting of our American Academy, has advocated the burr to this point and then abandoned it as it became the most useful. The round burr is now again brought into use, working from behind forward, removing the bone down to the level of the floor of the naris. A peculiar action of the burr is that the bone is removed, leaving the mucous membrane on the nasal side intact; thus bleeding is reduced to a minimum and the nose does not have to be packed while operating. Incision is now made with a small knife and the flap thus formed laid upon the floor of the maxillary antrum. The gauze pack is inserted through the nose and removed carefully the following morning. The entire time of the operation takes scarcely more than twenty minutes. There is no shock, practically no facial swelling, and the operation cures completely, no matter how chronically diseased the antrum may have been. A perfect drainage is secured and, after the third day, if it is found necessary, the patient may be given a syringe and taught how to cleanse the cavity.

It may be remarked, in passing that the Halle[4] frontal sinus operation is particularly adaptable to certain types; that the Kronlein operation is greatly hastened with electric equipment; and that the small burr takes out the button of bone with greatest facility in the intra-nasal dachryocystotomy or West operation. On account of the thin, fragile bone encountered, there is no indication for the burr in exenteration of the ethmoid sinuses. On the contrary, recent experience has demonstrated the technic of J. A. Pratt,[5] as published last year, to be the operation par excellence.

The operation in which a far greater number of otologists are using the burr, is, of course, the mastoid operation. This operation is particularly adaptable on account of the dense bone necessary to be removed. Inasmuch as the radical operation is practically a continuation of the simple, we will discuss the radical only.

Briefly, the technic is as follows: Under ether anesthesia, an incision is made with one sweep of the knife through the periosteum, exposing the bone. Hemorrhage is controlled by pressure through sponges held by the assistant, while the periosteum is rapidly elevated with an Andrews periosteotome. No haemostats are used, but hemostasis is at once accomplished by the immediate use of Jansen's retractors—an incidental saving of ten minutes operating time. The No. C burr is placed over the suprameatal triangle and a few seconds of application usually suffices to expose the mostoid antrum. Burrs F and G now remove the cortex. The small round burrs are found most useful in thoroughly exploring the accessory cells and completely exenterating the mastoid process. Stacke's director is placed in position and the small burr A used in removing the bridge. The elongated round burr, No. 1 or 2, is now brought into use and the annulus tympanicus removed, giving special attention to the hypo tympanicum. This can be done safely and quickly with the burrs and allows for easy and thorough after-treatment. Lack of thorough work at this point of the operation with the older instruments is, no doubt, one of the main reasons why so many cases were not completely cured. Another most important reason was the incomplete exenteration about dangerous areas. The eustachian tube is taken care of by the Robinson burr. A distinct advantage of this method of operating is the absolutely smooth cavity obtained, thus allowing healing to extend rapidly. No skin grafting is necessary. A thorough irrigation with 1 to 1,000 bichloride solution is used to remove bone dust and debris.

To those who have not been obtaining the good results expected, or, as one member of this society has recently put it, have been "becoming soured on the radical," this method of operating is recommended.

DISCUSSION

DR. J. A. PRATT, Minneapolis: I want to thoroughly endorse this paper and I do so by an experience of 14 years in the use of the electric burr in head surgery. My work was taken under Dr. Frank Allport, who was the first one in the United States to use the electric burr. He was using it at a time when most doctors were laughing at the idea of using

[3] Lemere, H. B. The Diagnosis and Treatment of Latent Antrum Disease. Trans. American Academy of Ophthalmology and Oto-Laryngology, 1919.
[4] Halle. Max. Frontal Sinus Operation. Archiv fur Laryngologie und Rhinologie Band 29, Heft 1.
[5] Pratt, J. A. An Ethmoid Operation. Trans. American Academy of Ophthalmology and Oto-Laryngology, 1919.

an electric burr. The instrument I use is the old Victor model and while it is just a matter of what we are used to, I think it has the best handle for the purpose. It has a straight handle and I use it just as I would a pen which gives absolute control. You can make it cut very lightly or by using both hands, make it cut very rapidly. You can burr most anywhere with it and work very rapidly, doing it with as little shock as burring a tooth.

When I speak about a simple mastoidectomy and letting the patient go home the next day, it is a simple mastoiditis with out complications. You are not in any place where you should have shock. In doing simple mastoidectomies with the electric burr, there is no shock, so that the patient can easily go home just the same as you would let a tonsil case go home. If we use ether we use very little, and generally we use gas in our operations. A man was talking to me today who had tried to do the operation after reading the articles, using a dental engine and burr. This is not suitable as it lacks power and the burrs are too small. It is best to see a man do the operation. I think at least every clinic week during the past three years we have had one case which shows how the burr can be used. You can take the covering off the sinus, you can open on to the dura and the shock is practically nil. We have two or three cases operated on in the morning, who went home at night—no more shock than you would have with a simple tonsillectomy.

Of course, there have been a great many things written on the kind of electric bone engines and it seems to me that the one you are familiar with gives the most satisfaction. They are not expensive, one case will pay for it. When once used you will wonder why you did not use it before. A simple mastoidectomy very seldom runs over twenty minutes, and the lessened work, the ease and freedom of using it, and the freedom from any particular danger or shock is really remarkable.

DR. H. I. LILLIE, Rochester: My personal experience with mechanically driven instruments is very limited, but in visiting clinics where these mechanical burrs are used, I have seen some very beautful work. The use of the burr in the hands of very experienced men in doing the radical operation is not without danger. The operation is not 100 per cent, in that it is frequently necessary to operate on the mastoid where you have not access to electricity. I prefer to use instruments which I can use at any time and in any place. I have no personal objection, but I think I would not attempt their use in preference to the usual instruments.

DR. L. W. MORSMAN, Hibbing (closing): There is little to add. I did not go into the operations very much in detail on account of lack of time. I want to cite a case of a little boy on whom I operated recently. I found an atypical sclerotic mastoid in which the seventh nerve came over and down into a split pea-sized antrum, covered only by a thin shell of bone. As the antrum was opened, the burr passed directly against the seventh nerve, uncovering it to the extent of five or six m.m. I would hate to think what would have happened to that nerve had I been operating with hammer and chisel. As it was, there was not even a traumatic paralysis and, when I went to see the little boy the next morning, he was sitting up with that "two-sided grin" that we always like to see in these cases.

I have never injured a lateral sinus with this equipment, and I have never seen one, or any other vital structure, injured with the burr. As to one hundred per cent operations, I think it is the general consensus of opinion that medicine and surgery has not been developed to that point of perfection.

As a former assistant to Dr. Joseph Beck, I very naturally fell into the use of using this equipment in head surgery. The technic, however, is simple and needs no special preparation. A great many men think that, because a quarter horse power, high speed motor, is used with resulting high speed of instruments, the equipment is dangerous. As a matter of fact, the opposite is true, as witness: the professional wood carver with a similar equipment, working out designs of minute detail. I might suggest to the new beginner that he have his old instruments laid on the side table. This I still do, as I am not sure of the electrical current in our locality, although it has not happened to fail me yet. I would no more think of doing, for instance a mastoid, without the aid of electrically driven equipment than I would consider doing a tonsillectomy under general anesthestia without the aid of an electrically driven ether vapor suction apparatus.

ACUTE LYMPHATIC LEUKEMIA*

GEORGE DOUGLAS HEAD, M. D.
Minneapolis, Minn.

Acute lymphatic leukemia, like many other diseases, presents itself in a variety of clinical symptoms difficult of interpretation without a study of the blood. Being a comparatively rare disease, the clinician frequently overlooks it because he fails to remember its important clinical features. Many a case of acute lymphatic leukemia has run its course and passed into the death records diagnosed as purpura hemorrhagica, scorbutus, purpura rheumatica, Werlhof's disease, typhoid fever, acute septicemia, diphtheria, or purpura simplex. If all the cases presented a well defined leucocytosis, the diagnostic problem would be much easier. Such is, however, not the case.

Studied from the standpoint of the blood, we

*Read before the Minnesota State Medical Association, St. Paul, October, 1920.

have two rather distinct types of the disease. One form exhibits a high leucocytosis, reaching even a figure as high as 550,000. In this type, the small mononuclears predominate. The other form shows a varied leucocyte count, depending often upon the stage of the disease when the patient first comes under observation. Some of these cases show a normal or a decreased leucocyte count in their early stages and a moderate or high leucocytosis before death. (Case VII). Other cases exhibit an early leucocytosis and a leucopenia before exitus (Case VI) while in the third group the count remains normal, or below normal, from the beginning up until termination (Case II). The last type to which I have referred is, however, rare. I have seen one such case (Case II) to which I shall refer later, which ran a leucocyte count between 5000 and 6000 throughout the whole course. This latter group of cases differs from the type with the high grade leucocytosis in the high percentage of large mononuclear cells.

The first of the two types (the high leucocytosis group) to which I have referred has long been recognized and, with the enlarged lymph glands, swollen spleen, well marked leucocytosis and the increase in mononuclears to 85 per cent or more, offers little difficulty in recognition. The other form, described under a variety of terms, such as acute pseudo leukemia, aplastic leukemia, alymphatic leukemia, aplastic anemia, etc., is not so clearly described by writers upon blood diseases, and the profession has been left in a confused state of mind relative to the distinctive features of this form of the disease. Of the two forms, to which I have called your attention, the latter is the more rapid in its onset and runs a course much like acute septicemia, with continued rather high fever, marked weakness, hemorrhages into the mucous membrane of the gums, stomach, bowels, and skin, a moderate degree of enlargement of the lymph glands and the spleen, a severe, rapidly developing anemia, and death, sometimes within a few days, but more often three to eight weeks. The enlarged lymph glands rarely become of sufficient size to arest one's attention and must be felt for, to be detected. It is a mistake for a clinician to wait for the appearance of enlarged lymph glands, visible to the eye, before considering lymphatic leukemia. Any acute infection with hemorrhages into the skin and mucous membranes, with or without enlarged glands, calls for its consideration and exclusion.

The first case of this kind which I can remember having seen was twenty-three years ago. The patient was a boy (Case I) about nine years of age. His continued fever of ten days standing and the enlarged spleen, persisent nosebleeds, with ill defined skin manifestations, suggested typhoid fever, and this was the early diagnosis. When the breath became foul and the gums bleeding, gangrenous and sloughing, diphtheria, scorbutus, and canerum oris were all considered, only to be ruled out. The boy died after an illness of three weeks and the autopsy showed no typhoid, diphtheria, scurvy, or malignant disease. At the time I could not classify the disease and, in the absence of blood findings, was not able to establish a diagnosis. In the light of my later experiences, I now unhesitatingly place the case in this group of acute lymphatic leukemia, of which I am now speaking.

In some of these cases with the low leucocyte count the diagnosis of typhoid fever is not infrequently made, the persistent temperature, bleeding at the nose, and enlarged spleen making a suggestive clinical picture. Some years ago I saw such a case with the late Dr. C. J. Ringnell (Case II). The patient was a young man, twenty-one years of age, with continued high fever of three weeks standing, with nose bleeds, enlarged spleen, and rapidly developing anemia. The diagnosis of typhoid fever seemed justified, with these exceptions,—the Widal reaction was negative and his differential leucocyte count showed 93 per cent of mononuclears, the large mononuclears predominating. This boy lived five weeks after the onset of his sickness, which was fairly acute. The enlarged lymph glands in this case were first noted above the clavicle at the end of the fourth week, and rapidly appeared in the groins, axillae, and neck. They were never large enough to attract one's attention. The autopsy revealed no lesions of typhoid fever. The postperitoneal lymph gland were of hen's egg size. The lymph glands of the axillae, groins and supraclavicular regions were enlarged, as were also those of the mesentery. The diagnosis of the pathologist confirmed the clinical interpretation of the case as that of acute lymphatic leukemia. This young man

gave a history of a severe septicemia, following an infection of his toe, one year prior to the onset of his fatal illness. From this, however, he had entirely recovered. In going over the histories of my own cases and those reported by other clinicians, I have been struck with the number in which some kind of an acute infection has preceded or occurred at the outset of the illness. For example, in Case XII, a carbuncle developed upon the back of the neck one month prior to the onset of his leukemia. Case I complained of toothache a few days before the gums began to bleed and his fever came on. In Case VI, the patient gave a history of having had a purulent discharge from the nose, of several weeks duration, about a year before he came down with his prostration and bleeding gums, and this nasal discharge had recurred with the onset of his leukemia. Case X began with acute tonsilitis and Case VII had had an ulcerated tooth removed before the onset of his sickness. This observation raises the interesting question as to whether or no some very virulent infective organism may not act upon the bone marrow to produce the overproduction of mononuclears, just as in a streptococcus and staphlococcus infection the bone marrow is stimulated to produce an increase of polymorphonuclear neutrophiles. However, blood cultures are negative. In Case II cultures before death and from the heart's blood sixteen hours after death produced no pathological organisms. Blood cultures have been made during life in Case III, Case IV and Case IX with negative results. In Case IV, however, the blood cultures after death showed a streptococcus pyogenes in heart's blood and in hemorrhagic lesions of the skin of the leg. The heart's blood also gave a gram positive coccus and two varieties of a gram positive bacillus.

In some of these very acute cases, the clinical diagnosis of purpura hemorrhagica, or pupura simplex, or, if pains are present in the limbs or about the joints, pupura rheumatica, may be made. In a boy of twelve years (Case III) which I saw with Dr. Annis, the purpura upon the legs led to the diagnosis of purpura simplex, and later, when vomiting of blood occurred and the stools became black and tarry, the diagnosis of pupura hemorrhagica was made. A well marked leucocytosis of 90,000, with a mononuclear count of 98 per cent established the correct diagnosis of acute lymphatic leukemia. This boy survived six days. He had no fever. A great subcutaneous hemorrhage, the size of the palm of the hand, appeared upon the left thigh.

In babies and very young children, these hemorrhagic manifestations in the skin, with or without fever, may present a clinical syndrome very difficult to differentiate from scurvy, without a blood examination. Such a case came under my observation about fifteen years ago (Case VIII). The patient was a child, six months of age, and had been sick seven weeks. There were numerous petechiae on the forehead and lips and under surface of the tongue. In the abdominal wall, just above the anterior-superior spine, was a black and blue hemorrhagic spot, one inch in diameter. The lymphatic glands of the axillae, groins, and neck were not palpable at first, but enlarged before death. The leucocyte count was 18,000, of which 90 per cent were nononuclears. Before death the child vomitted blood. The autopsy confirmed the diagnosis of acute lymphatic leukemia, but one feature of the bone marrow study deserves special mention, namely, the large number of eosinophiles. I had never seen such a crowding of the bone marrow with these cells before. It seemed to me that every third cell was an eosinophile. No similar case, so far as I know, has been reported.

There is no characteristic clinical picture in these acute cases with slight or no leutocytosis. The enlarged lymph glands may be suggestive but are often late in appearing and must be searched for by the clinician. Every case of purpura of any type, or persistent bleeding from the mucous membranes of the nose, gums, stomach, or bowels, associated with fever, and anemeia, calls for a careful examination of the blood to exclude or establish the diagnosis of acute lymphatic leukemia. It is the blood examination, with the presence of the high percentage of large mononuclears, way beyond the proportion seen in any other disease of which I have knowledge, which must be relied upon to establish the diagnosis beyond question.

A striking clinical feature in some of the cases is the ulcerative and necrotic lesions observed on the gums, sides of the cheeks, tonsils and pharynx. When the lesion first appears, one is impressed with the notion that he is dealing

with a severe, ulcerative type of diphtheria, or with mercuric stomatitis, or scurvy. As the diseased process advances, the ragged, deep, ulcerating, bleeding, crater-like lesions develop on the gums, sides of the cheeks and tonsils; the teeth become loose and the picture suggests to the diagnostician some rapidly developing malignant process like cancrum oris. Case I, previously referred to, presented in the later stages of the disease this clinical picture, and, in the recent case of one of our much beloved confreres blood smears from which were submitted to me for examination, the ulcerative, necrotic condition of the gums suggested trench mouth, and such was the diagnosis made early, prior to the systematic examination of the blood.

Sometimes these necrotic, ulcerative lesions center in the tonsils on one or both sides and throw the practitioner off his guard. In 1915 I saw such an instance (Case X). The man had been under observation for three weeks with the diagnosis of streptococcic tonsilitis. He had a foul breath, ragged, deeply ulcerated, gangrenous looking tonsils, enlarged cervical glands and a few petechial spots upon his face. His leucory count was 240,000 with 98.5 per cent mononuclears. The man died two days after he was seen.

In Case XI a male clerk 26 years of age whom I saw with Dr. W. H. Condit, the history of repeated attacks of tonsilitis preceded the onset of the nasal hemorrhages. The leucocyte were 42,000 with 85 per cent large and small mononuclears.

Not all the cases of the type of the disease without leucocytosis exhibit hemorrhages from the mucous membranes. I have had recently under observation a girl of twelve years, referred by Dr. A. T. Ridgway of Annandale, Minn., (Case IX) which presented in all respects the clinical symptoms and blood picture of acute lymphatic leukemia, but without hemorrhages from the mucous membranes or from the stomach or intestines. In this case the lymph glands were not sufficiently enlarged to attract one's attention, but, by palpation, they were from pea to almond size in the neck, axillae, and groin. Careful search for hemorrhagic areas in the skin and mucous membranes was negative, except for one or two pin-head size hemorrhagic spots on the soft palate. The complete blood examination in this case established the diagnosis beyond question. The leucocytes were 3,800, while the large and small mononuclears at various examinations ranged between 84 and 90 per cent.

A study of the clinical features in that form of the disease associated with high leucocytosis presents variations sufficient to keep the clinician upon his guard lest he fall into error. These cases as a rule run a slower course and more time is allowed for securing data and establishing conclusions which time does not modify. In some of these slow, persistent nose-bleed and bleeding from the gums is a conspicuous feature. In 1910 I saw with Dr. Walter Sheldon such a patient (Case VI). He had been ill about two months with fever and weakness. He was having severe and persistent nosebleeds and also some bleeding of the gums. His leucocytes were 35,000, of which over 75 per cent were of the mononuclear variety, the large forms predominating. He died three weeks later. A curious feature in this man's blood was the drop in leucocytes as the disease progressed, a few days prior to death, the count being only 7000. The development of multiple, raised nodules in the skin, lymphocytoma (Warthin) may be a conspicuous feature of some of the cases of this type. Some years ago I saw with Dr. John Butler a remarkable case of this form of the disease, (Case VII) which he has reported. The patient was a single woman of thirty-five years. Four months prior to my visit an ulcerated tooth had been removed leaving a bleeding socket, which did not heal well. From that time on she felt tired, had severe neuralgic pains through the shoulders and arms, and developed a little fever. Seven weeks after the onset, a raised nodule appeared in the skin of the flexor surface of the right forearm. A few days later a similar nodule appeared on the back. At the time I saw her, there were great numbers of these in the skin over the entire body. They were from pin-head to filbert size, had a soft, doughy feel, and were not tender to touch. Over the face they were so numerous as to create folds in the skin and give the lion-faced appearance of leprosy. Curiously enough, while the diagnosis of acute lymphatic leukemia was suspected in this case, at the time I saw her it could not be established by blood examination, since the

leucocytes were 3000 and the mononuclears, large and small, 28.5 per cent. Blood examination made in the later weeks of the disease presented the characteristic picture, with leucocytes rising to 52,000 and mononuclears to 96 per cent, the large forms predominating. This type of the disease is not so rare. It may run an acute or more chronic course. There were similar nodules in Case IV and in a case which I saw some years ago in the Minneapolis City Hospital service, a man forty-eight years of age, with fever and marked anemia, the notes on which are too fragmentary to report. There were many of these nodules composed of large mononuclear cells in the skin of the forearms and thighs.

In some cases, the skin lesions present a honey-combed surface with underlying necrotic areas. This was a conspicuous feature in Case XI. Over the hips and sacrum, pea to half dollar sized raised blebs first appeared. A few days later these became honeycombed with small openings leading into underlying necrotic, black looking ulcers in the corium.

Inasmuch as the hemorrhages, with or without lymphocyte infiltration, occur in other structures than the mucous membranes and the skin, remarkable clinical phenomena arise in the course of the disease. In Case XI, disturbances of vision and hearing occurred, probably due to hemorrhages into the sheathes of the auditory and optic nerves. In Case III, the right arm and leg were paralyzed, due to brain hemorrhages either into the ventricles or the arm and leg center upon the left side. In this case also the right eyeball was rotated outward and upward. Cases VIII and III vomited blood and passed blood by bowel, probably due to ruptured vessels in the stomach and intestinal wall. Case III passed bloody urine, while in a case seen with Dr. D. C. Cowles the notes of which are too fragmentary to report, there was bleeding from the vagina. Retinal hemorrhages were present in Cases II, III and VI. Some of the patients complained bitterly of pain along the course of the nerve trunks. In Case XIII pain and stiffness in the left leg was an early symptom. Case IV had severe persistent pain in the back and legs, Case VII a dull, heavy pain in the back of the neck and in the shoulders and arms. Case XIV had as an early symptom pain in both legs, Case XI had abdominal cramps, while in a man about forty-five years old, whom I saw with Turck in Vienna, a severe persistent headache ushered in the disease.

I have endeavored to present a clinical picture of the many manifestations of this remarkable disease which will, I hope, assist in its more general recognition. Since all the cases die within a few days or weeks, the correct diagnosis is of importance in order to establish the prognosis. The writer, from personal experience, can sympathize with the clinician who makes the diagnosis of scurvy or diphtheria or trench mouth or purpura simplex or purpura rheumatica, all diseases of relatively low mortality, and finds to his horror that the case is one of acute lymphatic leukemia ,with death only a few days or weeks away. The diagnosis is rarely absolutely established without the blood picture, including a differential leucocyte count. The clinical picture which the disease in full bloom presents is rather characteristic. As Bradford and Shaw state, having once recognized the first case, the others are readily interpreted. A number of times the writer has correctly suspected acute lymphatic leukemia, from a clinical study of the case, but never could summon the courage to state his conclusions positively without the knowledge which the blood examination could furnish. The cases without leucocytosis or with leucopenia resemble very closely aplastic anemia. The enlarged spleen and swoolen lymph glands in leukemia are the important points in differentiation. Those cases in which the lymph glands hypertrophy late in the disease, of which I have observed three, are very puzzling. However, attempts at such fine clinical differentiations are largely academic since the etiology of neither aplastic anemia or acute lymphatic leukemia is known.

Case I. Willie B., 10 years of age, male, schoolboy. Had been a weak child from infancy. On February 17th, 1897, while attending school, began to have a toothache and was sent home. A few days later he began having fever, the gums commenced to bleed, and an ulcer appeared upon the gums. Examination one week after the onset revealed a poorly nourished boy, waxy, pale skin, white mucous membranes with oozing, necrotic looking gums, pea to bean sized glands of the neck and submaxillary region. His temperature was 104, pulse 120. The spleen was enlarged to percussion, but could not be palpated. Liver not enlarged. The heart was nega-

tive. There were signs of an old, inactive tuberculous process in the left apex of the lung. No petechiae upon the skin. The boy was observed until his death eleven days later. He ran a high temperature, 102 to 105 degrees, throughout the course of his illness. A slow oozing from the nose and gums continued off and on until death. Considerable pain was complained of about the jaws and face. The breath was very foul. The gums were friable and necrotic, bleeding easily when touched. A purpuric rash appeared on the chest three days before death. The lymph glands of the neck were enlarged at the outset and those in the axillae and groins became enlarged before death. Bacteriological studies of the necrotic exudate from the gums and tonsils were negative for bacillus diphtheriae. The urine was negative except for albumen. Diazo reaction negative. Widal reaction negative. No blood studies were made. During the course of the disease, typhoid fever, septicemia, diphtheria, scorbutus, and carcrum oris were considered, only to be ruled out. The autopsy made twelve hours after death disclosed no adequate cause for death. There was an old healed tuberculous lesion in the left lung. A few petechial hemorrhagic spots were present over the pericardium and the pleurae. The post peritoneal lymph glands were enlarged, as also those of the neck, groins, and axillae. They presented the appearance of haemo lymph nodes. No pathological studies of the tissues were made. I regret that the incomplete clinical and pathological report of the case does not permit of a careful diagnostic differentiation. The similarity in the clinical features of the case to others reported later leads me to unhesitatingly classify it as one of acute lymphatic leukemia.

Case II. Mr. T., (Seen with Dr. C. J. Ringnell) twenty-two years of age, single, University student. Family history negative. Always been a strong robust young man. One year ago at the Freshman-Sophomore class fight was hit on the right foot with a piece of wood. The foot became infected and the boy nearly lost his life with acute septicemia. His recovery, however, was complete in about two month's time and he remained entirely well up until April 26, 1906. He began then to feel languid and his friends told him he was getting pale. He kept at his work for about one week and began to have nosebleeds. The bleeding was of a rather slow, oozing character. The first day he used eight or ten handkerchiefs. He then consulted Dr. C. J. Ringnell who found him with a fever of 99.8, slightly enlarged spleen, and a nose full of gummy, black clots. Typhoid fever was suspected and he was sent home and to bed for further observation. No rose spots, however, appeared. The Widal reaction was negative. The boy became very pale and anemic and continued to run a temperature between 99.5 and 102. The diagnosis of typhoid fever could not be established. Patient was seen in bed three weeks after onset. He was extremely anemic. His face had a puffy appearance. The whole body presented the lemon yellow appearance of pernicious anemia, the mucous membranes, being pearly white. There was some oozing from the nose of a pale bloody serum. No enlarged lymph glands could be palpated. The liver was palpable just below the costal margin. The spleen was enlarged and could be felt at the costal margin. Temperature 101. Examination of the blood showed the leucocytes 3000, red cells 2,050,000, hemoglobin 28 per cent, slight poikilocytosis, no nucleated reds. Differential: Small Monos. 76.9 per cent, Large Monos. 16 per cent, Polys. 6 per cent, Eosin. 0.0, one mast cell and two myelocytes seen in counting 355 cells. In spite of the absence of enlarged lymph glands, the diagnosis of aute lymphatic leukemia was made. Patient was observed for eleven days time until his death. Fever was continuous throughout, ranging as high as 105 towards the close. The slow oozing from the nasal mucous membrane continued. The spleen remained enlarged. About one week prior to death, the glands of the neck, groins, and axillae began to enlarge, but at no time during the course of his illness were they larger than from pea to bean size. Blood examination made four days prior to death showed the hemoglobin 25 per cent leucocytes 4000. Large Monos. 66.2 per cent, Small Monos. 32.4 per cent, Polys. 1.4 per cent, No eosinophiles, no mast cells, no nucleated reds seen. No hemorrhages into the skin were observed. No hemorrhages from the stomach or bowels. At the autopsy fourteen hours after death, the following were noticed: The skin was a lemon yellow color. A purpuric spot the size of a bean on the lower lip. Lungs negative except for hypostatic congestion at the bases. Heart somewhat enlarged. No valvular defects. An enlarged gland the size of a filbert was seen just below the greater curvature of the stomach in the omentum. Postperitoneal lymph glands were much enlarged and surrounded the aorta in a mass the size of a hen's egg. The mesentery lymph glands were enlarged, also those in the groins, axillae, and supraclavicular regions. The gastrointestinal tract was negative except in the cecal end of the ileum, where one or two superficial ulcers the size of a dime were noted. Stomach and pancreas were normal. Liver and spleen were enlarged, the spleen being twice the normal size. Both kidneys showed hemorrhagic areas at the cortices. In the liver two well marked, V shaped areas extending from the cortex toward the interior were seen. Bone marrow specimens taken from the ribs showed the presence of large numbers of marrow cells of the hyaline variety. Pathological diagnosis—acute lymphatic leukemia, hypertrophy of the heart, hypostatic congestion of the lungs, hypertrophy of the lymph glands of the mesentery, axillae, groins, supraclavicular regions, and postperitoneum, ulceration of the small bowel, purpura of the skin of the lip.

Case III. James P., (Seen with Dr. H. B. Annis) 18 years, single, student. Family history negative. No serious diseases as a child, although not very

strong. Never had tonsillitis or articular rheumatism. Three days prior to the onset of his illness he complained of feeling tired. On the morning of November 9th, 1917, after a fair night's rest, he noticed some dark blue spots upon his lower limbs. They were equally distributed over both legs below the knees. The next day he called the attention of his parents to the spots but was up and about the house. On that day his nose bled once or twice and he wiped some blood off from his gums. The spots had not increased in size. The third day a physician saw him and diagnosed purpura simplex. There was no fever and no vomiting. The gums bled again and also the nose. Urine passed looked smoky and contained blood cells microscopically. On November 12th a hemorrhagic area the size of a dollar appeared on the skin over the left hip. On the 13th he vomited blood. On that day he began complaining of his right arm and leg. He said they were partly paralyzed. He fainted on that day, going to the bathroom, and nearly lost consciousness. He was seen in consultation on that day. He was in a semi-stuporous state. Could be aroused enough to answer questions. There was some bloody mucous on the lips and slight oozing of blood from the gums. The right eyeball was rotated outward and the right pupil slightly more dilated than the left. The right arm and leg were completely paralyzed. There was no Babinski or Kernig. A fine purpuric rash was present on the legs from the knees downward. In the skin of the left hip was a large black hemorrhagic area nearly the size of the palm of one's hand. On the lower lip, a pinhead size purpuric spot was present, as also a half dime hemorrhagic area just below the suprasternal notch. A fine, half concealed purpura could be seen on the forearms. The vomited material contained streaks of blood. None had been present in the stools. There was no stiffness of the neck. Lymph glands of pea to bean size could be felt on either side of the neck, also above the left clavicle under the rami of the jaws, in the axillae, groins and epitrochlears. The spleen was enlarged and could be palpated just below the left costal margin. The liver could not be palpated. No masses were present in the abdomen. There was no fever. The leucocytes were 90,000, with a differential count of P. M. N. 1.5 per cent. Small Monos. 8 per cent, Large Monos. 89 per cent, Trans. 1.0. Eosinos. .25, Baso. 0, Myel. 0. 400 cells were counted. The clinical diagnosis of acute lymphatic leukemia was made. The boy died that day. Blood culture taken prior to death was negative. No autopsy was permitted.

Case IV. F. B., 32 years, male, laborer, married, three children. Admitted to City Hospital, Minneapolis, November 28, 1912. Family history negative. Denies venereal disease, except gonorrhea four years ago. Four years ago had some kind of a sickness with severe cramps in his stomach which lasted three or four days. A big lump appeared in the abdomen at that time which remained only a few days and then disappeared.

He was admitted to the hospital for marked weakness and raised lumps in the skin on various parts of the body. He stated that he began to feel sick about six weeks prior to admission. In the beginning he had diarrhea and pain in the abdomen. The nodules in the skin began to appear about one week later. They first appeared upon the legs, later upon the arms, then on the face and finally on the trunk. Six times in the last month he has had profuse nosebleeds. Three weeks ago he took to his bed on account of weakness. He had had no pain up to that time. Of late he has had pain in his back and legs. He does not know whether he had fever or not. His diarrhea has now stopped.

The physical examination reveals a large framed, broad shouldered man of stocky build. He is well nourished but shows a high grade of anemia in the pale yellow color of the skin and the white mucous membranes. There is bleeding from the nose and oozing from the gums, with black clots in the mouth around the gums. One's attention is at once attracted by the nodules in the skin. These are present all over the whole body, except on the hands and feet, and the exterior surfaces of the knees. They vary in size from seven to eight mm. They are raised above the surface of the skin and are of a soft, doughy consistency. They can be moved over the underlying tissues. They are most numerous over the chest and forehead, where they appear about one mm. thick over the thighs, abdomen and legs, and to a lesser degree over arms and forearms. Each nodule is surrounded by a purplish hemorrhagic zone one to 2 m.m. in diameter. Over the ventral surface of the chest and forehead many nodules show no hemorrhagic zone. On the first metacarpus of the right hand is a nodule thicker and firmer than the others. The lymph glands of the neck, groins, and axillae are palpable and of pea to filbert size. The physical examination was otherwise negative, except slight enlargement of the liver and spleen. Temperature 101 degrees. Pulse 102. The urine was negative. Blood showed R. B. C. 2,464,000, hemoglobin 29 per cent, W. B. C. 6,000. Differential count—P. M. N. 37 per cent, L. Monos. 31.6 per cent, S. Monos. 31.6 per cent, Eosino. .8 per cent.

During most of the time the patient complained of pain and stiffness in his back, neck and limbs. He had continuous fever, 99 to 101 degrees. He gradually grew weaker. On the fifteenth day in the hospital he passed three tarry stools and had a profuse nosebleed, lasting six hours. Toward the close he developed delerium and died on the seventeenth day in the hospital.

Summary of autopsy findings. (Dr. S. M. White, pathologist.) Skin—many soft raised nodules in the skin over the whole body except hands and feet. Abdominal cavity—postperitoneal and mesenteric lymph glands enlarged and hemorrhagic. Pleural cavities—fibrous adhesions on left side. Pericardial cavity contains 15 c. c. of clear serum. Three or four minute hemorrhages on diaphragmatic surface. Oesophagus

—negative. Thyroid—negative. Lungs—no area of consolidation; cut surface exudes frothy serum. Heart—epicardial surface near base shows numerous minute fresh hemorrhages; normal in size. Aorta—negative. Spleen—somewhat enlarged; pupil soft, purplish color; kidneys negative except that pelvis of kidney shows two or three hemorrhages in the mucous membrane. Liver—weight 2470 gms; slightly enlarged; adhesions over right lobe. Pancreas—negative. Stomach—a number of small nodules in the stomach wall near the pylorus and in the body. Bone marrow of ribs normal color and consistency.

Autopsy Diagnosis. 1. Purpura hemorrhagica with erythema nodosum. 2. Hemophilia. 3. Minute hemorrhages into the pericardium, epicardium, and mucous membrane of right renal pelvis. 4. Marked anemia. 5. General lmyphadenopathy. 6. Chronic perihepatitis. 7. Fatty degeneration of liver. 8. Acute splenic tumor. 9. Lymphoid nodules in gastric mucosa.

Bacteriological examination. Heart's blood a gram positive coccus and two varieties of gram positive bacilli and streptococcus pyogenes (?) Streptococcus pyogenes (?) obtained in pure culture from the skin of the leg.

Histological examination. Small intestine lymphoid hyperplasia. Liver—periportal connective tissue infiltrated with plasma cells. Adrenals—lymphocytes in medulla. Lymphnodes—sinuses packed with phagocytic endothelial cells. Skin nodules-infiltration with lymphoid plasma and phagocytic endothelial cells.

Case V. Mrs. L., (seen with Dr. H. M. Scheldrup) age 35, married, two children, Jewess. Family and past history negative up to two months ago. At that time, although previously strong, she began to feel tired and showed a disposition to avoid exertion. She had had no sickness that could account for her weakness. She became quite pale and was sent to a hospital but continued to grow weaker, paler and more prostrated. She could not sleep and had occasional vomiting spells. Her temperature ranged between 100 and 104 degrees. At this time her red cell count was 1,670,000, hemoglobin 25 per cent. White cells 16,000, with about 80 per cent large and small mononuclears. There were a moderate number of megaloblasts and normoblasts seen.

When first seen she was lying in bed, very weak and pale. She was fairly well nourished. The left nostril was packed with cotton to stop bleeding, which had started that afternoon. No bleeding of gums, no purpuric spots. One or two small lymph glands were palpable above the clavicles on either side. The only other abnormal findings were a soft systolic bruit heard at the apex and over the body of the heart, and a mass lying in the left lower abdomen, apparently arising from the pelvis and extending two cms. above the pubic arch. It was rounded and tender. Pelvic examination showed a hard indurated cervix and immovable uterus, but no distinct mass or fluctuation could be made out. Liver and spleen were not enlarged. No edema of legs or ankles. Blood examination now showed 32,000 leucocytes, 1,200,000 reds and 15 per cent hemoglobin. The differential count showed about 80 per cent mononuclears. Blood culture and urinalysis were negative.

Case VI. Mr. E., (seen with Dr. Walter Sheldon) age 40, married, one child. In the carriage business. Family history and past history negative. The patient had not been well for over a year, during which time he had had a great deal of business worry and hard work, entirely indoors. The winter before he was seen, he had been south and, while away, developed a severe nasal infection and was bothered for several weeks with a purulent discharge from his nose. About two months before the examination, he began to lose strength and grow pale in color. One morning, while dressing, he nearly fainted. He finally became so weak he went to bed. He had had considerable nasal discharge all this time and several severe attacks of epistaxis. The gums had bled once. The examination disclosed a fairly well nourished man, pale mucous membranes, and a lemon yellow color of the skin. He looked weak and was easily tired by talking. His temperature was 101. There was no discharge from the nose. Eyegrounds showed small hemorrhages in the retinae. The rest of the examination was negative except for three or four pin head sized purpuric spots on the inside of the right arm, and one or two the size of a dime below the knees. These were dark brown in color and looked like bruises in the skin. They were not over the shins. The leucocyte count, when first observed, was 35,000, with 75 to 80 per cent mononuclears. The blood examination at the time of my examination showed a leucocyte count of 15,000. Red cells 2,400,000. Hemoglobin 15 per cent. A differential count showed fully 90 per cent mononuclears, the predominating type being of the medium and large forms. The patient died three weeks later. Before death the leucocytes dropped to 7000.

Case VII. Miss R., (seen with Dr. John Butler) age 35, single, American, stenographer. Family history and personal history negative. Had never been sick before. Had weighed 135-140 before illness. About four months before the time this patient was seen, she had had an ulcerated tooth removed. The wound did not heal promptly, but bled a little for several days. From this time she began to feel tired and lose weight, although previously she had been a very active and indefatiguable worker. She continued running down and by the end of six weeks had begun to have a dull, heavy pain in the back of her neck. This pain gradually spread into her shoulders and arms and in one week had worked down to the waist. The pains became sharp and finally she became unable to move the arms, except with great effort and discomfort. The arms and hands felt cold, but perspired profusely. Temperature at this time was said to be over 100. At this time (7 weeks after onset of symptoms) a raised nodule appeared in the skin of the flexor surface of the right forearm. This was not red or painful, and although small at first,

steadily grew in size to about that of an almond. Shortly after another nodule appeared on her back, between the shoulders. During the following month, the pains of which she had complained, spread into the hips, legs and feet. Fever and loss of weight continued. No more nodules appeared in the skin until the end of the month, when numerous small tumors developed in large numbers on the face, then on the extremities and finally upon the trunk. Only the palms and soles were free from nodules by the end of a week. Since that time the nodules had steadily increased in size and number, and loss of weight had been progressive. The only favorable sign was a cessation of the pains in the limbs, except for a very few attacks. Her knee joints and some of her finger joints became swollen and stiff. She was greatly bothered by night sweats. Had never had nose bleed.

Upon examination, a tall, thin, intelligent looking girl was seen lying in bed, dreading to be moved. Pupils reacted to light. Knee jerks present. The skin presented a most remarkable appearance. Over the entire body were scattered enormous numbers of soft, doughy, raised nodules, varying in size from that of a pin head to two cm. in diameter. Over the face the large number of nodules threw the skin into folds, thus simulating the Lion Face of Leprosy. Many of these, especially on the lower extremities had a bluish color. Sometimes these became more pinkish in color, the patient stated. The nodules were not tender to touch and were not distributed especially along nerve trunks. Careful examination failed to reveal any swelling in the long bones or lymph glands, and the liver was not enlarged. The spleen was just palpable at the costal margin. No masses to be made out in the abdomen. Heart and lungs negative.

Blood examination showed, Hgbl. 55 per cent, R. B. C. 2,750,000. WBC. 3000. Differential P. M. N. 67.0. Large Lymph. 3.5. Small Lymph. 26.0. Trans. 0.5. Eosin. 2.0. Turck's stim. 1.0 per cent. Two megaloblasts seen in counting 200 cells. Anisocytosis, poikilocytosis and polychromatophilia were present in moderate degrees.

A section taken from one of the nodules was diagnosed non-pigmented sarcoma of the skin. Urine showed a very faint trace of albumin, otherwise negative.

During the succeeding five weeks the patient's condition rapidly became worse. Emaciation became extreme, hearing became greatly impaired and she could not speak above a whisper. The nodules had increased in size and blood taken at this time gave the following picture: Hgbl. 24 per cent. R. B. C. 1,900,000. WBC. 52,000. Differential: P. M. N. 3.5. Large Lymph. 91.5. Small lymph. 4.5. Trans. 0.5 per cent. One normoblast and one megaloblast seen in counting 200 cells. Pockilocytosis, anisocytosis, and polychromatophilia marked.

Case VIII. Baby N., eight months of age, female, a well child until six weeks prior to the time she came under observation, when the parents noticed a change in color of the child's skin to a waxy white. She became restless, weak, developed fever ranging between 99.5 and 101. Numerous petichiae appeared on the forehead and on the lips. These were followed by hemorrhagic spots on the dorsum of the tongue and the buccal mucous membranes. On the abdomen, over the right anterior superior spine of the ileum a large black and blue spot the size of a quarter appeared. Many small, pinhead sized purpuric spots later came out on the skin of the abdomen. The liver and spleen were both palpable about one finger's breadth below the costal margin. At first no enlarged lymph glands were noted, but one week prior to death bean sized glands could be palpated in the neck on either side, in the groins and in the axillae. The temperature upon the seventh day of observation reached 105.4, dropping to 101 before death. The child vomited blood and passed blood in the stools three days before death. The leucocytes were 18,000. P. M. N.'s 8.85, Large Monos. 4.73, Small Monos. 85, Myel. 1.43 per cent, a few microcytes, macrocytes, poikilocytes, and one normoblast were seen in counting 200 cells. The clinical diagnosis of acute lymphatic leukemia was made. The child passed into an asthenic, markedly anemic state and died after an illness of seven weeks. A complete autopsy was not allowed. No gross pathological lesions were found in the heart and lungs except fine, pinpoint hemorrhages in the pleurae and pericardium. The spleen and liver were enlarged, also the postperitoneal lymph glands. A specimen of bone marrow taken from a rib showed large numbers of mononuclear cells and many eosinophile cells. The presence of the eosinophile cells in such large numbers in the bone marrow I had never seen before. Every third or fourth cell seemed to be an eosinophile. The pathological examination confirmed the clinical diagnosis.

Case IX. Nannie G. (Referred by Dr. A. T. Ridgway) 12 years of age, Scandinavian. Family history negative. Diphtheria at eight years of age. Influenza in the winter of 1919. Father states the girl has looked white all summer and has seemed lanquid and weak and has complained of some aching pains in the limbs. Has had no nosebleeds or bleeding from the gums or any spots in the skin. Five weeks ago Dr. Ridgway was consulted, who found the patient had considerable fever, temperature 100, and a well marked anemia, and enlargement of the spleen and liver.

Examination today, Sept. 4, 1920, shows a rather thin, poorly nourished child. Color of the mucous membranes of the skin shows marked anemia. Physical examination negative aside from fever, temperature 100, marked anemia, a few purpuric spots upon the soft palate, enlarged lymph glands in the neck on both sides, also in the axillae and in the groins. These are from pea to almond size. The liver is palpable two to three finger's breadths below the costal margin. Spleen is palpable one finger's breadth below the costal margin. The heart is en-

larged, left border of cardiac area of dulness being one cm. outside the nipple line. There is a systolic blowing murmur at the apex area, transmitted to the axilla, with an accentuated pulmonary second sound. Blood pressure 98 systolic, 70 diastolic. Urine negative. Blood—hemoglobin 24 per cent, Reds 1,056,000, Leucocytes 3,800, P. M. N. 13.5, Small Monos. 53.0, Large Monos. 31.5, Trans. 1.5, no eosino., no baso., myelocytes 0.5 per cent, slight polychromatophilia, some poikilocytosis, no nucleated reds seen. Wassermann negative. Blood culture negative. Blood examination made Sept. 8th showed leucocytes 3,500, P. M. N. 9.0, Small Monos. 57.5, Large Monos. 33.0, Trans. 0.5 per cent. Clinical diagnosis acute lymphatic leukemia. The subsequent history of the case is not known.

Case X. Mr. H., age 35, single, Jewish. Family history negative. Patient had always been well. He had lues when 26, for which he received treatment.

At the time when first seen he had been sick about three weeks, during which time he was believed to have a streptococcus sore throat. When examined, his tonsils were seen to be badly infected, edematous and shiny. The cervical glands on both sides were enlarged. His temperature was 103.5. He was at times irrational. A few subcutaneous hemorrhagic spots were present on the face and nose. No other signs of organic disease could be detected. Acute lymphatic leukemia was was suspected and the blood examined. It showed 65 per cent hemoglobin, 3,800,000 red cells, 240,000 leucocytes. Differential: P. M. N.'s 0.0, large lymphocytes 38.0, small hymphocytes 60.5, unclassified 1.5 per cent. No transitionals, eosinophiles or basophiles. Patient grew rapidly weaker and died in coma two days later. No autopsy was permitted.

Case XI. Mr. B. (seen with Dr. W. H. Condit) age 26, single, mail clerk. Family history and personal history negative, except for frequent attacks of tonsillitis and nosebleed years ago. About that time he began to get weak and had to give up work and go into the country for four or five months. Felt better after the rest and went back to work. He became weaker and more prostrated and three months later gave up work entirely. During the past year he had had some disturbance of his stomach and had been pale, but had no other distinctive symptoms, up to three months ago. At that time ecchymotic spots had appeared on both ankles and he was running a fever. The prostration became severe and the man continued to grow paler and temperature higher, and six weeks before the examination he had had several severe hemorrhages from the nose. The nosebleed became more frequent and required packing to control the hemorrhages. His condition became grave.

Upon examination, a pale, greatly prostrated young man was seen. He complained of disturbance of vision and hearing. There was a purpuric rash over the back, and necrotic areas varying in size from a pea to a half dollar were present over pressure points, hips and sacrum. These had appeared first as blebs over the skin, which had broken, and underneath a superficial necrosis of the skin had taken place. Small holes had punctured through into the subcutaneous tissue in various places. They were not bedsores. The liver and spleen were enlarged, each reaching one finger's breadth below the costal margin. There was slight enlargement of the cervical lymph glands, and also the axillary, most marked on the left side. These glands were isolated and not tender. No other enlarged glands. A complete physical examination was not made on account of the patient's weak condition.

The blood examination had shown 2,300,000 reds, 44 per cent haemoglobin, and 42,000 white blood cells, of which 85 per cent were of the large and small mononuclear type. Cultures from the blood and from the blebs were negative for organisms.

Case XII. Mr. J., aged 36, married, Scandinavian. No children. In good health and at his work as a contractor until three or four months before, when he seemed to lose energy and was tired all the time. About one month before the examination a carbuncle formed on his neck. This was lanced and soon healed. However, the patient did not gain but continued to get weaker. A few weeks later he had a severe nosebleed. Following this he became weaker and went to bed. He continued to have frequent and severe attacks of epistaxis and his anemia became marked. His temperature ranged between 100 and 102. His appetite was poor. He had no diarrhea. When the patient was seen he was in bed and having nosebleed, and his gums had been bleeding for several days. His pulse was rapid and his temperature 101.5. He looked very sick.

Examination showed a greatly prostrated, anemic man. No purpura. Lymph glands in groins and in left axilla slightly enlarged. Liver and spleen not enlarged. Further examination was negative outside of the blood. This showed a leucocyte count of 19,300. The differential count showed large mononuclears 70, small mononuclears 6, P. M. N. 18, myelocytes 1.5 per cent. There were a few nucleated reds of all sizes. Polychromatophilia was present. The mononuclears were not of the extremely large type, but of moderate size. In many of them the nuclei filled up practically the whole cell, only a thin rim of protoplasm being visible. Some nuclei showed vacuolation. A few basket cells were seen. The protoplasm of many of the large mononuclears had a tinge of pink and a very fine basophilic granulation. The nuclei stained a deep or paler blue.

A diagnosis of acute lymphatic leukemia, rapidly fatal, was made. The patient died about two weeks later.

Case XIII. Martin C. (referred by Dr. A. T. Ridgway) three years of age, male, American. Two brothers living and well. Father and mother well. A breast-fed child. Never sick until the present ailment. Three weeks prior to the examination (December 21, 1917) the baby began to complain of pain and stiffness in the left leg. The mother stated

that he seemed to be sore along the shin. The next day he got stiff all over except his arms and could not walk. After a few days the stiffness subsided, but the child began to get very pale in color. His color had always been good before that. While the child was able to get about, he kept crying about his legs hurting and had no appetite. The legs did not swell or become paralyzed. For the past two weeks the child has been about the same, except that his color is getting whiter. He has had a number of nosebleeds and once he vomited blood, which the mother thought he had swallowed. On physical examination, the child looked well nourished but very anemic. The skin had a slightly yellowish tinge. He seemed very listless and lanquid. There was some bleeding around the gums. The lymph glands of the axillae, groins, and neck were palpable, but none of them larger than a bean. The spleen was markedly enlarged, the liver palpable one to two finger's breadths below the costal margin. There were no hemorrhages in the skin. There was some tenderness and swelling over the tibiae on both sides.

The urine was negative. The Wassermann reaction negative. The blood showed R. B. C. 1,336,000, haemoglobin 15 per cent, W. B. C. 260,000. Differential count—P. M. N. .25, L. Monos. 7.5, S. Monos. 92, Trans. 0, Eosinos. 0, Baso. 0, Myel. .25 per cent. Color index 0.56. No megaloblasts or normoblasts found. A diagnosis of acute lymphatic leukemia was made. The child was not seen again.

Case XIV. John K. (referred by Dr. Schroeder of Maple Plain, Minn.) Male, 4 years. German descent. Father living and well. Mother had anemia but is now well. Three brothers living and well.

The child had never been sick until about two months ago when he began to complain of pains in his legs. About four weeks after the onset of the pains in the legs, a purpuric rash appeared upon the legs which was preceded by an attack of vomiting. At this time the child developed temperature, which was normal in the morning but ranged as high as 101 degrees in the afternoon. The rash upon the legs lasted about ten days. The child drank a great deal of water but had no appetite. The bowels were constipated. He had lost much in weight and and had grown feeble and weak and very pale in color. The purpuric rash appeared again upon the legs a few days prior to the examination. He had bled some from his nose at various times, but none from the gums or bowel.

The examination revealed a poorly nourished child with a very waxy white color of the skin. The child was so weak as to remain entirely indifferent to the examination, with its eyes closed. One or two small purpuric spots were present on the left arm just above the wrist and also on the back of the left hand. A dime sized black and blue spot was present over the left ankle. The spleen was enlarged, reaching one finger's breadth below the costal margin. The liver was palpable about three cm. below the ribs. A few lymph glands of pea size could be felt in either axillae and on either side of the neck. No masses were palpable in the abdomen. No tenderness or roughening over the long bones. The urine was negative except a trace of albumen and a few hyaline casts. The blood picture was typical of lymphatic leukemia. Haemoglobin 15 per cent, R. B. C. 700,000, W. B. C. 26,250. Differential leucocyte count showed about 94 per cent mononuclears, large and small. The child died the day following the examination.

DISCUSSION

DR. J. P. SCHNEIDER, Minneapolis, Minn.: It is very seldom that we have an opportunity of gathering together fourteen cases of acute lymphatic leukemia. I think Dr. Head is to be congratulated and we all are to be congratulated on his list of cases showing the various forms which this disorder may take. After all, the type picture of acute lymphatic leukemia is met with but seldom, the aberrant forms being much more common. The great difficulty lies in the fact that we are dealing with a complex, the etiology of which is unknown. The safest criterium, as Dr. Head so well pointed out, is the blood picture very carefully studied but it does not suffice to ascertain the total lymphatic white cell proportion. Having 95 per cent of the cells of the large lymphocyte form is not sufficient. The point is, are they pathological cells and how high is the proportion of pathological cells, because we must always remember that there is a possibility of error in differentiating acute lymphatic leukemia from acute lymphadenitis which does occur and in which the count may be as high as 80,000, and in which 90 per cent will be large lymphocytes, and yet the patient recovers. Dr. Turck mentions five such cases in which he says that in a certain part of Austria he lost his reputation, for the patients all reovered. So it is plain to us that the proportion of pathological cells is what matters. This is particularly true in children in whom the lymph adenoid ring is involved.

The next phase which the Doctor pointed out is that at times the local condition is prominent, and in such conditions as these the glands may become larger and harder and more pronounced, and frequently the blood picture recedes, but the reverse is also true.

I think the location determines a great deal. If it occurs in the nose, throat, gastrointestinal tract and so on the out-swimming is easy, but there are places where the out-swimming is not easy. Occasionally it is found principally in the spleen; occasionally retroperitoneal, and finally there is an out-swimming which will be pronounced.

Another point which Dr. Head did not bring out is the early help we can get by an ophthalmoscopic examination. In a patient seen not long ago with supposed pernicious anemia the ophthalmoscopic examination gave me the clue that it was an acute or subacute leukaemia. The findings are highly characteristic.

DR. H. Z. GIFFIN, Rochester, Minn.: I am very

much interested in the timely presentation of this subject. There is no doubt that the diagnosis of cases of acute leukaemia is frequently missed. The cases concerning which there is most confusion are those which simulate acute septicemia and acute ulcerative endocarditis. Cases with acute stomatitis present themselves before a typical blood picture has developed. I remember one patient who came with cerebral hemorrhage in whom the diagosis was easily made from the blood picture. There has recently been under observation a patient with clinical symptoms suggestive of acute leukemia in whom the blood picture was not typical, even just preceding death. The absence of extreme leucocytosis is not rare. On the other hand, we see cases of simple pyogenic infection associated with leucocytosis and a high lymphocyte and mononuclear count which are not leukaemia; the blood picture later becomes normal.

I would like to emphasize the importance of a careful study of the blood smear. A study of the morphology of the blood renders relatively easy in most instances the recognition of immature forms and stem cells, upon which a diagnosis depends.

DR. E. L. TUOHY, Duluth, Minn.: I rise to discuss this paper briefly because during the past winter I saw in the space of one month three cases typical of lymphatic leukemia. One case started out with what was taken to be severe sciatic pain. This patient evidenced at no time severe hemorrhages, but went on with rapid prostration to death.

The second case began with bleeding, suggesting an ordinary purpura, with hematuria, bleeding from the bowel, and developed a severe condition of the gums.

The third case I saw when the patient was about to die and in that individual there had been a lesser degree of hemorrhage, chiefly from the bowel.

I am impressed with what Dr. Schneider just spoke of, the necessity of studying these calls as to their pathological imprint: In all of these instances the cells were unusually large, the nucleus deeply strained and nearly filling the cell with only a lesser outer ring of faintly staining protoplasm. And by that same token I am led to go back to a certain case seen with Dr. Rowe some years ago that had a very large percentage of large lmyphocytes, but I do not recall whether they bore the pathological imprint or not. The child was alive some months later.

In view of the fact that I saw three of these cases in such a short time it occurred to me that there might be some connection with the epidemic of flu that we had just passed through.

DR. L. A. NIPPERT, Minneapolis, Minn.: We are certainly under obligations to Dr. Head for bringing this very important subject to us, because it is a disease that is not at all characteristic. It has to be hunted for and the abnormal features are the ones which are most puzzling. Furthermore, the establishment of the disease means an absolutely fatal prognosis. So many different manifestations will mislead you, as the Doctor has mentioned, and, as he also mentioned, one of our beloved confreres recently died of this disease. I will take the liberty of relating this case because it is one of great interest. The Doctor had been south and came back with a sore mouth, which was diagnosed as Vincent's; the spirilla were found. When I saw him he had this condition and a leukopenia, the white cells showing 3,000. The question was whether it could be a case of Vincent's bacilli with this low blood count. Then the differential count was made and it was shown that 90 per cent were large lymphocytes. With this finding there was no question but that the prognosis was fatal. There was no enlargement of the glands and very little of the spleen. His mouth was swollen like a stomatitis; there were no hemorrhages and the fever was of an irregular type. The leuocytes finally increased to 12,000, of which 90 per cent were large lymphocytes.

From the few cases I have seen I am more impressed with the fact that when anyone has symptoms of sepsis an examination should be made to exclude this disease.

DR. J. W. ANDREWS, Mankato, Minn.: I have always recognized the difficulty of diagnosing acute lymphatic leukemia, and as I hear the discussion of these men, all of whom live in the cities and have great opportunities for diagnosis, I am further impressed with the difficulties the country practitioner is under in diagnosing this fatal and very important disease.

As to the blood picture, Dr. Head has told us, and others, that there may be a very high leucocytosis and that there may be a leucopenia. In typhoid we surely get leucopenia and in appendicitis and other infectious diseases a high leucocyte count. What are the symptoms, I ask myself as I hear these gentlemen discussing the disease, what are the symptoms that the country practitioner is going to rely upon to get a diagnosis? I want you all to recognize the difficulty under which the country practitioner is laboring in diagnosing these obscure diseases. He does not make a blood count and without the blood count and the differential count he is much at sea in his diagnosis. Dr. Head tells us that sometimes the lymph glands are enlarged and sometimes not, and that it is in the latter part of the disease that they are enlarged. I would like to ask Dr. Head if it is not true that in the late cases of lymphatic leukemia this condition exists, would not a careful examination in the early stage reveal enlargement?

I have nothing to add to the splendid paper and splendid discussion we have had. I was about to make a motion that Dr. Head be given further time, for this is very important, probably more important than any other subject that will come before this meeting. I shall be glad if he may be given a little more time to clear up some of the points that are so obscure to the country practitioner, and I should also like to hear a little more from the country physicians.

DR. MOSES BARRON, Minneapolis, Minn.: Just a

short time ago I had an opportunity of studying at autopsy a child 2½ months old who had presented peculiar symptoms. About two weeks before death the patients eyes became reddened, swollen, temperature ranged between 101° and 104°, and swellings developed at the inferior maxillary regions on both sides. Small reddish nodules appeared over the body. The physician who was called could not make a diagnosis.

At the autopsy there were found nodules over the chest and abdomen, three to five mm. in diameter. The peritoneal lymph nodes were markedly enlarged, and tumor masses were present in both kidneys. At first, I was not entirely certain of the diagnosis of these masses, but microscopic study revealed their true character. It proved to be a case of lymphatic leukemia with infiltration into all the viscera including the corium of the skin. The entire trachea was also infiltrated with lymphoid cells. The nature of the lesion had not been recognized during life. No blood examinations had been made before death.

In the case that Dr. Nippert spoke of which was reported by Dr. Head, I examined some of the slides. though at first there was a leucopenia the leucocytes gradually increased in number to 12,000. An interesting feature in this case was the character of the cells themselves,—large lymphocytes rather embryonic in type with nucleoli present with in the nuclei of most of them. The presence of nucleoli within the nuclei of lymphocytes is an important factor in establishing the fact that the cells are abnormal in the blood and approach the "stem" cell type.

The invasion of the skin in leukemia is found more often in the chronic types of the disease. There was a chronic case recently at the University Hospital which showed an enormous involvement of the skin. Several hundred tumors were scattered through the skin, ranging from 3 mm. to 3 cm. in diameter. All of the palpable lymph nodes were also greatly enlarged. The histologic picture of the lymph nodes and the masses in the skin was that of leukemic infiltration. However, the blood picture was entirely normal; there was no increase in the number of leucocytes and the differential count was not altered. Some of the lymphocytes appeared abnormal in structure. The patient died within a short time after reaching the hospital, and at the time of death there was no leucocytosis present or anything at all pathological in the differential count. The diagnosis at the time of death was aleukemic lymphadenosis.

I wish to emphasize the importance of studying the blood for immature cell types in suspected cases of leukemics and also the relative frequency of infiltrations into the skin in these diseases.

DR. GEORGE DOUGLAS HEAD, Minneapolis, (closing the discussion): I appreciate how crowded our program is and the Chairman was very generous with me in the time allowed for reading the paper, so that I will not consume much time in discussing the interesting points which the discussion has brought out.

I was glad that Dr. Tuohy spoke of the pain in the course of the nerve trunks in one of his cases. That was brought out in the part of the paper which I left out. A number of cases gave very severe pain, one in the jaw, another in the arms and shoulders, and others in the legs. Some of these required morphia for relief. The pain was evidently due to hemorrhage into the nerve sheaths. A case in point that I saw in Vienna with Turck was that of a man with persistent and severe headache, so severe that it required large doses of morphine to relieve. The man died in about two weeks from the onset of his leukaemia. There are many interesting features that we have no time to discuss.

One thing in relation to the point which Dr. Schneider brought up I would like to speak about. Of course, to a man who is experienced in blood work and to those who are examining blood smears all the time, the large pathological mononuclears may come into the foreground and be easily recognized, but for the average man who does not often make a differential blood count, I doubt if he would get much comfort in attempting to differentiate pathological mononuclear cells from those he sees in the normal blood. Such interpretations, I think, would have to be made by men who are working all the time in blood work. But I am very sure that when any one of you has once seen and recognized one of these cases of acute lymphatic leukaemia, the clinical features will be so striking that you will at once insist upon someone examining the blood for you, if you cannot do it yourself, whenever another case, clinically simulating the condition, is met with. There is something very suggestive about the clinical syndrome when once a case has been seen. I thank you.

Physicians Income Tax

By A. H. Remole, Auditor of Physicians and Hospitals Supply Co., Minneapolis. Minn. Formerly Division Chief Internal Revenue.

Income tax returns of the physician presents difficulties that are not met with in ordinary lines of business. Personal expenditures often overlap business disbursements and the distinctions drawn in this and other expense items are so often confusing that in his desire to avoid error the tax payer generally pays a larger amount than is necessary. Some of the following suggestions may seem so obvious as to be unnecessary, but an examination of physicians' returns has disclosed that probably a large majority err in not taking advantage of at least a part of the suggestions made.

Many professional men still report the gross amount of business done, in lieu of the actual cash collected for the year from professional services. The Internal Revenue Bureau permits the cash method and it is by all means advisable as few doctors have the time to keep books on a complicated accrual basis.

Expenses incurred in attending medical conventions and clinics may be deducted as a business expense. This would include transportation cost in full and the cost of subsistence and lodging in excess of what it would cost at home. Such traveling expenses are also allowed in all cases where the physician is called out of town in his practice.

Subscriptions to medical journals and magazines kept in the waiting rooms for the entertainment of patients are considered a cost of doing business and may be deducted. This applies also to professional society dues and to commercial club memberships. Fraternal society dues are not included unless membership is held solely for business reasons.

Where an office is maintained in the home it does not usually occur to the taxpayer that this is a business expense. The government so considers it, however. If the home is owned, a proportionate share of depreciation, light, heat and servants' salaries may be deducted. A rented home receives the same privilege, except that a proportionate share of the rent is taken in lieu of depreciation. For example, one room used as an office in a ten-room house would entitle such a taxpayer to one-tenth of the costs named.

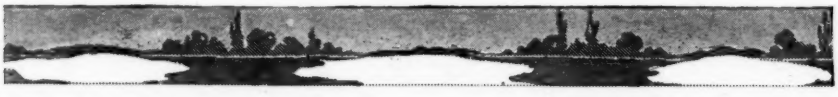
Telephone and telegraph costs run into considerable money in a years time. Some physicians take advantage of this permissible deduction, but very few consider the expense of maintaining the home phones. These are also considered as a business expense determined by the approximate business usage.

The physician probably uses his automobile for business a larger proportion of the time than any other class. The Income Tax Laws and Regulations permits a deduction in an amount determined by the amount of time the car is used professionally. Depreciation, gasoline, oils, storage, tires and minor repairs are the principal expense items allowed. Due to the hard usage that is given a doctor's car, a fair rate of depreciation is 25 per cent. A $2,000 car used four-fifths of the time for business would entitle the taxpayer to four-fifths of $500 as deduction for depreciation.

So much decrepancy is found in the amount of materials used by physicians having approximately the same professional income that one must conclude that full advantage is not always taken of these expense items. Gauze, drugs, antitoxins are only a few of such expenditures. Almost anything of this nature may be deducted. Such costs must not, however, include new instruments, furniture and books or other articles that are not actually consumed during the year. Instruments of such a nature as to be easily broken or lost may be included with other materials, however. Depreciation may be charged on instruments, books and furniture. This charge off may generally be computed at about five per cent for furniture and books and ten per cent on instruments. X-ray and similar machinery depreciates much more rapidly than instruments and a larger rate of depreciation may therefore be taken. If your experience shows that a certain electrical machine is gone in three years, take 33 1-3 depreciation on the cost.

On account of the rapid strides forward of the medical profession, it often happens that books and instruments become obsolete. In all such cases the Revenue Department will permit the taxpayer to charge off the original cost of such an asset less depreciation sustained.

In view of the fact that a physician need only report the cash taken in during the year as income, the Income Tax Regulations do not permit such a taxpayer to charge off as expense work done gratis, nor to charge uncollectible professional accounts. Collection expenses, which would include legal fees, postage, stationery and printing may, however, be taken.



MINNESOTA MEDICINE

Owned by
The Minnesota State Medical Association.

PUBLISHED BY ITS EDITING AND PUBLISHING COMMITTEE

R. E. FARR, M. D. Minneapolis
H. LONGSTREET TAYLOR, M. D St. Paul
L. B. WILSON, M. D. Rochester
F. L. ADAIR, M. D. Minneapolis
J. T. CHRISTISON, M. D., St. Paul

EDITORIAL OFFICE
CARL B. DRAKE, M. D., EDITOR
1300 Lowry Bldg., St. Paul

BUSINESS MANAGER
J. R. BRUCE, 403 Central Bank Bldg., Saint Paul
Telephone: N. W. Cedar 1683

All correspondence regarding editorial matters, articles, advertisements, subscription rates, etc., should be addressed to the Journal itself not to individuals.
All advertisements are received subject to the approval of the Council on Pharmacy and Chemistry of the American Medical Association.

Subscription Price: $3.00 per annum in advance. Single Copies 25c
Foreign Countries $3.50 per annum.

Vol. IV March. 1921 No. 3

EDITORIAL

JUSTICE IN MAL-PRACTICE SUITS

The present legislature has at the present writing two bills for its consideration which are of great importance to the medical profession of the state.

The one, reducing the statute of limitations from six to two years is familiar to the profession throughout the state and is obviously just. That a patient is able under the interpretation of the present law to sue a doctor for alleged mal-practice for services rendered as long ago as six years seems obviously unjust and lays the profession open to an unnecessary and unfair liability. Some judge, some time ago, interpreted the law, however, and this is justification from the law standpoint.

The second bill is the more important and, in brief, aims at a furtherance of justice abstractly and justice to the defendant doctor in particular. According to the present law, under what is known as Statutory Privilege all information acquired by a physician from his patient is considered strictly confidential and is not admitted as evidence in court. In our February issue we called attention to the desirability of preserving in general this confidential relation between physician and patient. However, when matters of a private nature are made public in court by a patient in a mal-practice suit against a medical man, all reason for preserving the confidential nature of the individual's history is abolished. To withhold medical testimony which might be obtained from a physician who had subsequently or previously or even in conjunction rendered services to the patient and which might give a truer picture of the case either for or against the doctor sued, is unjust. It will be argued that physicians, through a certain esprit de corps, will testify to the disadvantage of the patient. It is not typical of the medical profession to perjure itself and we believe the alteration in this law would work for a furtherance of justice.

It is startling to note some statistics recently gathered by a prominent medical protective insurance company. These show that in 1918, 1 to 4 per cent of mal-practice suits instituted against the profession exceeded $5,000.00. In the following year, 1919, the percentage increased to 35 and in 1919 the percentage jumped to 55. Do these statistics indicate that mal-practice is on the increase? Certainly not. Depreciation in money value of course accounts for it in some degree and perhaps the unsettled condition of the country in general comes in for its share. It may be taken, however, as an indication for the profession to bestire itself and support with considerable zest legislation directed toward the attainment of justice for itself.

THE LATE OSTEOPATHIC BILL

The Minnesota legislature was only one of several state legislatures which had to consider granting the osteopaths the right to practice major surgery. We have every reason to believe that the question will be raised in the next legislature.

Osteopathy, founded upon an empirical assumption as to the cause of disease, has gradually broadened the scope, not only of its training in the fundamental medical sciences, but in its therapeutic activities. When an osteopath wants to do major surgery, however, and place the public at the mercy of doctors with much poorer qualifications than the average graduate of the medical schools (and every physician knows these are meager enough), it is up to the

medical profession and all others informed, to act for the welfare of the public.

This bill was obviously a short-cut to surgery and if passed, osteopathy would have attracted a certain type of man who is constantly on the lookout for short-cuts and the chance to get something cheap.

The remedy for what promises to be a continual tilt between medical sects, lies in the establishment of standard requirements for those practicing the healing art, be he surgeon, physician, dentist, osteopath, chiropractor or chiropodist. If an osteopath wishes to be a surgeon, let him meet the requirements.

Dean Lyon of the Minnesota Medical School, has called attention to the practicability of the State Board of Education passing on the eligibility in the fundamental sciences of candidates desiring to practice the healing art. Or, in Minnesota, where we have just one medical school, he believes the university may logically exert this function.

The soporific conditions of the State Medical Association, induced by the recommendations of the last legislative committee, received a rude awakening by the osteopathic bill. Some constructive legislation by the profession is in order to prevent a repetition of this recent experience with such dangerous legislation.

CASE TAKING

The progressiveness of the Southern Minnesota Medical Association has been shown by an action at its last meeting, which attracted but little attention at the time—the appointment (to be formally voted upon at its Winona meeting next summer) of a Committee on Clinical Research, whose duty it shall be "to endeavor to establish systematic methods of case taking among all the members of the Association and particularly among those who are in general practice. This committee will also act in an advisory capacity to members of the Association who are encouraged to refer to them questions pertaining to the above clinical data."

This action follows the recognition of what Sir James Mackenzie points out in Oxford Medicine—that the early stages of disease have not been given the attention they ought to receive, and that "until symptoms are classified on a basis having reference to the principles underlying their production, medical progress will be delayed."

That the logical case taker is the general practitioner is evident. He it is who meets the patient first and who first realizes that the layman, though he may use the same words as the profession, may speak a different language. The clinician has been using for a varying number of years terms of definite scientific meaning; the layman has been accumulating a strange and marvelous melange of medical misinformation resulting in a distorted and nebulous vocabulary elusive of definition, for the interpretation of which the general practitioner in time compiles a glossary of pseudo-medical terms for each of his clientele.

In the examination of histories it frequently becomes apparent that not enough thought has been given to the underlying meaning of patients' expressions. If we examine more closely into the beginnings of disease, we note at the outset that subjective symptoms practically always precede objective signs and hence the words of the patient, who is usually a layman, furnish the important information. A closer and more critical scrutiny of the patient's words, than is usually given, is called for. It is a factor that enters constantly into the accuracy of histories. No word should be accepted at its face value; it is safer to consider all words descriptive of the body or its functions, when employed by the laity, as having a doubtful meaning until given a clean bill of health by one having intimate knowledge of the patient's peculiarities of diction.

Investigation is difficult enough with the patient, harboring symptoms and giving verbal evidence of their existence, in our presence, but where we are once or more removed, either in the person of the patient, in lapse of time or both, our difficulties multiply. Internal evidences of erroneous elements in the anamnesis appear in many case records.

In many cases the patient may appear to be well posted in the terms used and, should he be on a level with or, perhaps, above the level of the clinician intellectually, recognition may not be had that such a layman is not an expert in this line of knowledge and that his words,

phrases, and partial diagnoses require close dissection.

As the primary articulate source of information is the individual patient, it is necessary either to learn what meaning should be attached to the apparently significant words he uses or to teach him the correct use of scientific terms. As Dr. William H. Smith says: "Pain must not be the reflection of the surgeon's or internist's idea of pain, but what the patient means by pain must be determined."

The work of the committee will be one of great significance in the progress of medicine and the general practitioner—erstwhile thought to be moribund—will take on a new lease of life with this new task which will indeed try his mettle.

H. B. A.

WHITE SOLIDARITY

The world is at present dominated in all its commerce, government, science, by the white race. The maintenance of this commanding position depends on white solidarity. Such conditions of suspicion hatred and armed strife as have been occupying the world during the past few years, have kept the white races divided. A single, unsupported white nation might be overthrown by a restless one of another color.

If the Russo-Japanese war be considered in its significance as an inter-race strife, and study is given to the numerical strength of the various races, then some idea of the future possibilities of white overthrow may be gained.

White solidarity will never be secure so long as the various nations continue to compete with one another in armaments. Such rivalry for military and naval supremacy will not aid the white race in understanding one another, or having such commercial rivalry and economic unity as is necessary for solidarity and continued domination.

The tremendous cost and gigantic burden of taxation which would have to be carried by the nations in a competitive scheme of armaments would soon bankrupt the world. It would be better to limit armaments by international understanding and to make sure of racial unity, than by an excessive burden of taxation to endanger the peace which the armaments plan to secure.

Would it not be better, instead of spending 90 per cent of the world's income on armaments and less than 1 per cent on health, to turn these figures about, and trust that through the improved health of the people, a vast measure of protection would be afforded? Words fail to express the enormity of criminal foolishness displayed in the past by many nations in the enormous amount of energy spent in the prevention and cure of disease, only to raise better physical specimens for slaughter.

Are we, as a nation, not continuing to do much the same thing? We are very active in a multitude of public health activities, both for the prevention and cure of disease. The sum spent annually is insignificant when compared with, for instance, the naval construction plan in progress.

Improved health, limited armaments, some revised form of a league of nations will be sufficient to ensure to the white race its continuation of world domination.

C. E. S.

NEWS OF THE HOSPITALS

Dr. F. L. Durgan, of Winnebago, has been appointed superintendent of the sanitarium at Nopeming, near Duluth.

The Board of Directors of St. Lucas Hospital, Faribault, has under consideration the plan of enlarging the building to double the present capacity.

Dr. Herbert O. Collins, superintendent of the General Hospital, Minneapolis, from 1909 to 1918, has been reinstated as superintendent, following the resignation of Dr. Walter E. List.

For conceiving and taking a large part in the work and building of the State Hospital for Crippled children, St. Paul, Dr. A. J. Gillette has been honored by the patients of that institution with the presentation of a full length oil painting of himself, done by W. W. Churchill of Boston. The fee of the artist was paid by the earnings of the older patients who work in the shop of the Phalen Park institution.

OBITUARY

DR. W. E. CHAPMAN

Dr. W. E. Chapman, Litchfield, Minn.; Vermont State University, Burlington, Vt., 1879; member Minnesota State Medical Association; died Feby. 5, 1921.

DR. J. C. FARMER

Dr. J. C. Farmer, McKinley, Minn.; University of Minnesota; member Minnesota State Medical Association; died February 9, 1921.

DR. L. A. WARD

Dr. L. A. Ward, Bemidji, Minn.; University of Illinois, 1895; aged 55; died January 16, 1921.

DR. IRA LESLIE EDMUNDS

Dr. Ira Leslie Edmunds, Clearwater, Minn.; University of Michigan, and Rush Medical College, 1884; member Minnesota State Medical Association; died January 2, 1921.

DR. FREDERICK R. MOSSE

Dr. Frederick R. Mosse of Rochester, Minn., was born at Sun Prairie, Wisconsin, November 1st, 1854, and died December 26th, 1920, in Rochester.

In 1869 he entered the Wisconsin University at Madison, where he graduated in 1874. In the same year he entered the Homeopathic Medical College of Chicago, received his diploma in 1877, and began practice in Rochester.

He was married in 1881 to Miss Flora L. Hurd, who, with one son, Frederick, survive him.

Thus for over forty years Dr. Mosse was a practicing physician in Rochester and Olmstead county. He was, for a time, a member of the board of pension examiners, health officer of the city, county coroner and physician, and a member of the library board.

"Dr. Mosse was known to those close to him as a refined gentleman. The number of intimate friends was not many, the doctor being too busy a man in his useful life to mingle as freely as some other citizens, but the strength of the friendships felt for him was all the stronger because of this fact. He served the public wherever he found it possible and wherever his abilities gained an outlet. As a professional man, he was everything that ethics demand. More than that, he was the doctor of no particular class. When the well to do called him, he was ready, but just as ready was he to respond to the call from some humble home where suffering existed, and scores and scores of times did he make such calls, knowing full well that there never could be any remuneration for his services. No kind of weather held him back. The quality of self sacrifice was large in him and if a man is remembered by the good that he does, the memory of Dr. Mosse will long be green in the community."

REPORTS AND ANNOUNCEMENTS OF SOCIETIES

MINNEAPOLIS CLINIC WEEK

Minneapolis will hold its fourth annual Clinic Week beginning April 25, 1921. The clinics will be conducted much the same as in previous years, with a special effort at coordination of the medical and surgical sides. Diagnostic methods will be given special emphasis.

Clinics will be conducted daily until three o'clock each afternoon, and the afternoon program will begin at four o'clock. Minneapolis physicians will appear on the program between four and five P. M., and at five o'clock a visiting physician of national reputation will give a discourse.

The Oto Laryngolic Society will hold its banquet during clinic week, and the Hennepin County Medical Society has chosen this week for its annual banquet. The visiting physicians will speak on each of these occasions.

The attendance at Minneapolis Clinic Week has shown a marked increase from year to year, which is indicative of the interest shown by the physicians of the state.

OF GENERAL INTEREST

Dr. P. E. Anderson, of Chicago, is now associated with Drs. Jacob and Benson in the Union Clinic at Willmar.

Dr. J. D. Lewis has been appointed chief of the eye, ear, nose and throat service at the Minneapolis General Hospital.

Dr. N. O. Pierce, of Minneapolis, entertained at a stag party for Phi Beta Phi Fraternity on the evening of February third.

Dr. J. A. Myers, of Minneapolis, has moved his offices from 1035 Metropolitan Bank Building to 323-327 LaSalle Building.

Dr. Verne S. Cabot, of Minneapolis, is in Chicago studying with Beck Brothers, where he is doing special work in surgery.

Dr. L. J. Kennedy, of St. Paul, has recently associated himself with Dr. Bohland of Belle Plaine, and will practice medicine at that place.

Dr. and Mrs. H. S. French, of Milaca, have moved to their new home at Grove City, where Dr. French will engage in the practice of medicine.

Dr. Hugh S. Willson, of Minneapolis, expected to return to his office about the middle of February from a short vacation spent in California.

Dr. Earl H. Phillips, of Austin, has left for Des Moines, where he will serve as an interne in the Taylor Clinic at the Des Moines General Hospital.

Dr. E. T. Reeve who has been doing surgical work

at the Sievertson Clinic in Minneapolis recently returned to Elbow Lake to resume his practice there.

Dr. McRae who left Rochester in February, 1920, to enter the American Red Cross service, has returned to the Mayo Clinic to resume his work there.

Dr. W. R. Humphrey, of Stillwater, has recently moved into his new offices on North Main Street, which he is occupying with Dr. Victor C. Thompson.

Dr. W. G. Palm, announces the opening of his offices in Crookston, Minnesota, about March first, where he will limit his practice to eye, ear, nose and throat.

Dr. Walker, of Wykoff, has recently sold his practice to Dr. H. W. Christianson, of Minneapolis, and has gone to Boston to spend this year studying and doing hospital work.

The Annual Congress of the Medical Education, Licensure, Hospitals and Public Health, will be held March 7, 8, 9, and 10, 1921, in the Florentine Room, of the Congress Hotel, Chicago.

Dr. and Mrs. R. H. Sweetman, of Menahga, are spending a few weeks in California. Dr. Peter E. Peterson, of St. Paul, is taking care of Dr. Sweetman's practice while he is away.

Dr. Alexander B. Moore, roentgenologist of the Mayo Clinic, Rochester, has been named regional chairman for Minnesota for the University of Virginia Centennial Endowment fund.

Dr. W. J. Dowswell of Bellingham, has left for Milwaukee where he will spend a year taking a special course in eye, ear, nose and throat disease. Dr. E. G. Ahrens will take over Dr. Dowswell's practice.

Dr. J. E. Soper of the United States Public Health Service and Dr. Harry Ritchie, of St. Paul, were guests of the Upper Mississippi Medical Society which held its annual meeting at Brainerd recently.

Waverly, Minnesota, is without a physician since Dr. Keene left to enter army service. There is a splendid opening at this place and any one interested should communicate with the Commercial Club of Waverly.

Dr. L. A. Walker, of Asbury Hospital, announces the following appointments on the attending and consulting staff: Doctors J. F. Corbett, Paul F. Brown, J. F. Avery, A. T. Mann, J. F. Smith, Olof Kittleson and G. F. Schmidt.

Dr. C. W. Woodruff, who has practiced medicine for the past sixteen years at Chatfield, Minnesota, and for the past year has been taking a vacation, will resume his practice and has opened offices in the old postoffice building.

Dr. S. R. Maxeiner, of Minneapolis, has removed his offices from 1035 Metropolitan Bank Bldg., to 323-327 LaSalle Bldg., where he will occupy offices vacated by Dr. Gilbert J. Thomas who recently moved his office to the new Nicollet Clinic.

Dr. M. O. Oppegard and Dr. O. E. Locken, formerly of Madison, Minnesota, are now connected with the Northwestern Clinic, which was recently organized at Crookston. They have equipped an office building of twenty rooms to take care of the various departments.

Dr. Harry J. Klein, of Duluth, surgeon for the police department for the last six years, has tendered his resignation and will engage in private practice. Dr. Joseph H. Cosgrove, for a number of years associated with a physicians' clinic at Duluth, has been appointed to fill the position.

Dr. C. F. Morsman, former resident of Hibbing, and a brother of Dr. L. W. Morsman, of Hibbing, has been elected president of the Spokane Academy of Medicine, a medical society which includes only specialists in medicine. Dr. Morsman is recognized as one of the leading specialists in Washington.

Dr. James Ewing, Professor of Pathology of Cornell Medical School, will give the annual Alpha Omega Alpha address March 10th, at 8:00 p. m., in the ampitheater of the Anatomy building of the University. His subject is "The Place of Pathological Anatomy in Modern Medicine." The public is invited.

Dr. Walter Courtney of Brainerd, Minn., resigned to the Crow Wing county board as a member of the sanatorium board of the Crow Wing and Aitkin counties sanatorium at Deerwood. The commissioners offered a vote of appreciation and thanks to the doctor, and expressed the regret that the condition of his health makes it necessary for him to withdraw.

Dr. John E. Soper, for many years a prominent physician of Norwood, Minnesota, who entered the World War and served with distinction, has been appointed inspector general of hospitals for disabled former service men for the district including the states of Minnesota, North and South Dakota, Montana and Iowa. Dr. Soper has been a resident of Minneapolis since his discharge from the army.

The Division of Venereal Diseases, of the State Board of Health, wishes to announce the opening of another evening dispensary under the cooperation of Wells Memorial House and the State Board of Health. This clinic will be open Wednesday and Saturday evening at 7:30 o'clock at Wells Memorial Dispensary 116 No. 11th St., Minneapolis. During 1920 the clinics in Minneapolis, St. Paul, Duluth and Virginia, operated in conjunction with this Division of the State Board of Health, admitted 1648 cases of venereal diseases and administered 32,604 treatments.

The Clinical Club of Minneapolis held its February meeting at which Dr. Moses Barron reported a case of lead poisoning and a review of the literature. Dr. Max Seham reviewed literature on vaccination and presented numerous statistics which it would do well for all of the medical profession to have at hand to refute the usual arguments of the anti-vaccinationists. The annual election of officers took place at which Dr. E. L. Gardner was elected president to

succeed Dr. S. R. Maxeiner, retiring. Dr. Clifford Boreen was re-elected vice president. Dr. J. Warren Bell was re-elected secretary-treasurer. Dr. W. A. Fansler and Dr. Kenneth Phelps were unanimously elected as new members.

Dr. E. O. Giere of Watertown, S. D., has accepted the position of chief of staff of the St. Paul Hospital, tendered him by the directors of that institution. He succeeds Dr. E. Boeckmann of St. Paul, and will assume his new duties about May 1st, removing with his family from Watertown to St. Paul. Dr. Giere has made rapid advancement in his profession and is recognized as a leading surgeon, not only in his own state, but throughout the Central West. Commencing his work at Hayfield, Minn., he later removed to a larger field at Madison, Minn., establishing a hospital there, and thence to Watertown, S. Dak., where the hospital which he serves as chief, has developed into a large and successful institution.

(Extracts from letter of Secretary S. M. M. A.)

At the Mankato meeting of the Southern Minnesota Medical Association it was recommended that the Committee on Clinical Research consists of tht following members: Dr. H. Z. Giffin, Rochester, and Drs. Walter Henry Valentine, Tracy and S. M. White, Minneapolis.

The Executive Committee says: "The Committee on Clinical Research is not to begin officially until after the next meeting, although they have been appointed and are going over the ground preparatory to beginning their work at once, after that time."

Dr. L. F. Fisher, of Thief River Falls, has received appointment as local surgeon for the Minneapolis, St. Paul and Sault Ste. Marie Railroad company.

Dr. L. M. Cruttenden, of Northfield, has gone to New York City to take a postgraduate course in aural surgery at Columbia University. He expects to be away for two or three months, after which time he will return to Northfield to resume his practice.

Dr. E. W. Benham, who has been doing post graduate work in San Francisco and Chicago for the past few weeks, has returned to Mankato, Minnesota, where he will resume his practice in the Mankato Clinic.

Dr. F. Marx White, professor of medicine at the University of Minnesota, has been appointed chief of the department of medicine by the Board of Regents, which also accepted the resignation of Dr. W. J. Mayo, as vice president of the board. Dr. Mayo, in resigning as vice president, did not sever his connection as a member. He was succeeded by M. M. Williams, of Minneapolis. Dr. White succeeds Dr. L. G. Rowntree, who has become associated with the Mayo Clinic at Rochester.

DeGraff, Minnesota, is greatly in need of a physician. This is a good locality and affords an opportunity to build up a splendid practice.

NEW AND NON-OFFICIAL REMEDIES

During January the following articles have been accepted by the Council on Pharmacy and Chemistry for inclusion in New and Non-official Remedies:

Calco Chemical Co.:
- **Cinchophen Tablets.**

Hynson, Westcott & Dunning:
- **Globules of Benzyl Benzoate.**

Heyl Laboratories:
- **Acriflavine.**
- **Proflavine.**

Intra Products Co.:
- **Calcium Cacodylate—IPCO.**

Winthrop Chemical Co.:
- **Salophen.**

Morgenstern & Co.:
- **Salophen.**

Neocinchophen.—The ethyl ester of methyl-phenyl-quinolin-carboxylic acid. It was first introduced as novatophan. The actions and uses of neocinchophen are the same as those of cinchophen (New and Non-official Remedies, 1920, p. 224), only it is tasteless.

Tolysin. — A brand of neocinchophen complying with the N. N. R. standards. It is supplied in the form of a powder and as tolysin tablets 5 grains. Calco Chemical Co., Bound Brook, N. J.

Saligenin.—Salicyl Alcohol.—Saligenin is a local anesthetic, similar in action to procaine. It is said to be as effective as procaine but much less toxic; also the anesthesia produced lasts longer, and for this reason the addition of epinephrin is not necessary. Saligenin is a white solid soluble in water.

Salicaine.—A brand of saligenin complying with the N. N. R. standards. Calco Chemical Co., Bound Brook, N. J. (Jour. A. M. A., Jan. 8, 1921, p. 113).

Pneumococcus Vaccine No. 14 — Beebe. — A pneumococcus vaccine (see New and Non-official Remedies, 1920, p. 285) containing Types I, II, III and IV diplococci pneumoniae in equal proportions, suspended in physiological solution of sodium chloride, each Cc. containing 500 million killed bacteria. Marketed in vials of 6 Cc., 10 Cc., and 20 Cc. Beebe Laboratories, Inc., St. Paul, Minn.

Typhoid-Paratyphoid Vaccine No. 39—Beebe. — A typhoid vaccine (see New and Non-official Remedies, 1920, p. 291) marketed in packages of three 1 Cc. vials, each Cc. containing 1,000 million killed typhoid bacilli, 500 million each of killed paratyphoid bacilli A and killed paratyphoid bacilli B, suspended in physiological solution of sodium chloride; also marketed in 30 Cc. vials. Beebe Laboratories, Inc., St. Paul, Minn.

Colon Vaccine (Acne) No. 11—Beebe.—A colon bacillus vaccine (see New and Non-official Remedies, 1920, p. 282) marketed in packages of six 1 Cc. vials, each Cc. containing 1,000 million killed colon communis bacteria suspended in physiological solution

of sodium chloride; also marketed in packages of one 10 Cc. vials and in packages of one 20 Cc. vials. Beebe Laboratories, Inc., St. Paul, Minn.

PROGRESS

Abstracts to be submitted to Section Supervisors.

MEDICINE

SUPERVISORS:
F. J. HIRSCHBOECK,
FIDELITY BLDG., DULUTH.
THOMAS A. PEPPARD
LA SALLE BLDG., MINNEAPOLIS

THE USE OF A HIGH FAT DIET IN THE TREATMENT OF DIABETES MELLITUS: L. H. Newburgh and Thurlow L. Marsh (Arch. of Int. Med., Dec., 1920). The authors state that the high protein, low fat, low carbohydrate diet, as usually given in diabetics according to the Allen regime, if given in sufficient quantities to maintain the needs of the body, shows an accompaniment of glycosuria in the severe cases of diabetes. In order to prevent this the caloric intake must be reduced so that inanition results, as an increase in the fat elements would immediately induce an acidosis. The physician is left with the alternative then of either having the glycosuria persist or have the patient suffer from inanition, due to the low caloric intake. The authors endeavor to solve this dilemma by revolutionizing the present conception of the treatment, by permitting a liberal fat diet, adding only enough proteid to maintain the nitrogen equilibrium, and a minimal amount of carbohydrates. They have adopted a more or less routine procedure as follows:

When the patient enters the clinic he is at once given 900 to 1000 calories, of which about 90 grams is fat, 10 grams protein, and 14 grams carbohydrates. After the patient has been sugar free for one or two weeks his diet is increased to about 1400 calories, of which 140 grams is fat, 28 protein, and 15 or 20 grams carbohydrates. With a larger person, a second increase is subsequently made after another period of trial, so that they are given 1800 calories, with 170 grams of fat, 30 to 40 grams of protein, and 25 to 30 grams of carbohydrates.

They have studied the effects of this treatment on 72 cases of true diabetes mellitus, with no selection of cases, the majority, however, being of the severest type. In spite of this latter fact, however, they have succeeded in rendering and keeping every patient sugar free up to the time of discharge.

They report 3 severe cases, in which the patients became promptly sugar free with the institution of their diet of 900 to 1000 calories, but who became promptly glycosuric again after the resumption of the high protein diet, later on again becoming sugar free by returning to their original diet. They add further that in no case did a serious acidosis develop.

Four of the 72 patients died in the hospital, but none of their deaths were due to their diet, they claim. One entered with Influenzal pneumonia; another was transferred from the surgical clinic, suffering from a severe sepsis accompanying mastoid disease; both patients dying within 24 hours after admittance. The third patient came to the hospital in coma and died ten days later. The fourth patient refused to limit herself to the diet, and went into coma after eating a bag of oranges.

Four examples are given of cases in which the acidosis promptly improved with their treatment, when it was present on entrance.

As a support to their belief that the protein intake can be considerably reduced, they cite the observation of Hindhede that less than .66 grams of protein per kilogram of body weight is required to maintain the nitrogen balance in healthy individuals, in the presence of sufficient calories from other sources. The diet of the authors is based on this observation. They realize that this may not be true in diabetics, however, and they have computed the ratio between the nitrogen intake and output by the usual procedures. They find that Hindhede's rule holds good in diabetics as well as in normal people.

Lastly, they show that the strength and capacity for work of the patient is astonishing, which they claim tends to prove that their diet is satisfactory in enabling their patients to lead an active life for an indefinite period.

In this first paper, which concludes with an appendix of diabetic diets as used by the authors, they claim that their treatment fulfills the four requirements of a satisfactory diet, namely, the prevention of acidosis, the maintenance of the nitrogen equilibrium, the absence of sugar, and finally, making it possible for the individual to resume the ordinary activities of life.

F. J. HIRSCHBOECK.

FIRST INFECTION WITH TUBERCULOSIS BY WAY OF THE INTESTINAL TRACT: Eugene L. Opie (Amer. Rev. of Tub., Vol. 4, No. 9, page 641). Autopsies made upon 93 children and 50 adults in the city of St. Louis showed no case of healed mesenteric tuberculosis. Among British soldiers in France, however, caseous or calcified mesenteric nodules were found in 18 instances among 66 autopsies. In 15 other autopsies on British soldiers the proportion of mesenteric tuberculosis was approximately the same. The greater frequency of tuberculosis among cattle in Great Britain is suggested as an explanation for the numerous cases of mesenteric tuberculosis.

Focal tuberculosis of the lungs was not found in a single autopsy that showed mesenteric tuberculosis. In two instances, however, healed apical tuberculosis occurred and in one there had been active widespread tuberculosis of the lymphatic system. Opie points out that the available evidence indicates that the first infection with tuberculosis does not uniformly

prevent a second infection but modifies its course so that it tends to become chronic and exhibits little tendeny to become disseminated. He also states that first infection with tuberculosis may occur by way of the lungs or by way of the gastro-intestinal tract; and the occurrence of one lesion tends to prevent the other. J. A. MYERS.

THE EFFECT OF ARTIFICIAL PNEUMOTHORAX ON PULMONARY TUBERCULOSIS IN THE RABBIT: H. J. Corpe, Saling Simon, O. B. Rensch (The Ameri. Rev. of Tub., Oct., 1920). Although artificial pneumothorax is a thoroughly established therapeutic measure, study of it in connection with experimental tuberculosis has been neglected.

The effect of compressing a diseased lung is generally accepted as being inhibitory to the tuberculous process. The increased fibrous tissue formation is best explained as resulting from the lymph stasis.

The question of the circulation in the compressed lung interested the authors, not only because of the paucity of literature but also because of the diversity of opinion and results from the few experiments which have been done. The conclusions drawn by Shaw in 1919 was the particular stimulant to the authors' research because he found that after intravenous injection of tubercle bacilli into the rabbit, the collapsed lung was found to be tuberculous when other organs were normal. Such observations, if true, would contra indicate pneumothorax especially in early cases and probably in all.

Rabbits were used in the experiments described. Intravenous injection of a uniform suspension of virulent human tubercle bacilli was done after a one side pneumothorax or hydrothorax had been performed. Controls were used, no compression being produced in them. The animals were killed 10, 19, 28 and 37 days after infection. They showed no difference in number or type between the tuberculous lesions in the collapsed and normal lungs. The result differs from that of Shaw who concluded that compression favors the development of tuberculosis in the compressed lung. In animals which were given suspensions of Prussian Blue, Scarlet R and starch, intravenously, the collapsed lungs showed a distribution of the dyes equal to the normal lungs. The gradual disappearance of the Prussian blue occurred uniformly on both sides which showed that the circulation was neither lessened nor improved in the collapsed lung. EVERETT K. GEER.

PULMONARY FINDINGS DUE TO CIRCULATORY CHANGES: J. S. Prithard and M. A. Montensen (Amer. Rev. of Tub., Oct., 1920). The authors point out that diagnosis of pulmonary tuberculosis is sometimes made and the patient subjected to an antituberculosis regimen over a long period of time when no clinical tuberculosis exists, but the physical signs and clinical symptoms are due to a disturbance of the circulatory system. For example, fibrotic changes in the lungs may accompany mitral stenosis and in such cases hemoptysis not infrequently leads to confusion in differentiating between circulatory deficiency and pulmonary tuberculosis.

Infarction of the lungs may cause such extensive pulmonary changes that the condition is confused with pneumonia. Again in some cases of extreme cardiac enlargement or pericardial effusion expiratory sounds, bronchial in character, due to the underlying compressed lung tissue may be heard over the back of the left base thus making it difficult to differentiate between pericardial or pleural effusion and pneumonia.

Bronchitis not infrequently accompanies cardiac lesions and when one is in doubt as to its etiology the administration of digitalis often causes marked improvement thus indicating that the circulatory deficiency is at least an associated factor in causing the symptoms. In many cases with bronchial symptoms such as are present in protracted colds, circulatory degeneration should be considered as a possible causative factor as well as upper respiratory affections and pulmonary tuberculosis.

From the above facts the authors conclude that all pulmonary signs and symptoms must be very carefully studied from the etiological point of view and that physical signs in the lungs should not be the only basis in making a diagnosis. J. A. MYERS.

TUBERCULOSE PULMONAIRE ET CANCER PRIMITIF DU POUMON: (Pulmonary tuberculosis and primary cancer of the lung) Maurice Letulle (Presse Med., August 7, 1920). The author reports two cases of combined pulmonary tuberculosis and primary carcinoma of the lung. In both cases cancer developed in the presence of active tuberculosis.

The first patient entered the hospital for advanced tuberculosis. Physical signs revealed abscesses at the right upper lobe and the patient was coughing up profuse purulent sputum containing acid fast bacilli. The tuberculous infection was not unusual, but the patient complained of excessive pain in the right side. During the course of the disease rather unusual symptoms developed. There was edema of the face; the veins of the chest, upper thorax, and right arm became distended. A diagnosis was made of obstruction to the superior vena cava. Autopsy findings showed the superior lobe of the right lung a cancerous mass containing many small tuberculous cavities which were almost obliterated by the cancerous ingrowth. Microsopic examination of the lung tissue showed epithelioma. The right pleura and mediastinal and cervical glands showed cancerous involvement and the superior vena cava was compressed by a mass of fibrous malignancy.

The second case was diagnosed at necropsy. The lung was filled by a caseating mass containing giant cells. Foci of definite malignancy were found in the bronchi. The alveolar spaces were also filled with large deeply stained cells containing prominent nuclei, many of which showed active cell division.

Cancer secondary to old tuberculosis is perhaps not rare. The epithelium of the bronchi and alveolar spaces can undergo cell division and become malignant in the same way that the other tissues in the body undergo malignant changes. Letulle states that his two cases with the combination of primary cancer and tuberculosis is a much rarer condition than the association of a metastatic cancer and tuberculosis. Metastasis to the lung and pleura, either by emboli or by direct extension is quite common.

SURGERY

SUPERVISORS:
E. MENDELSSOHN JONES
LOWRY BLDG., ST. PAUL.
VERNE C. HUNT,
MAYO CLINIC, ROCHESTER.

REPORT ON TETANUS: A. P. C. Ashhurst (Arch. Surg., 1920, 407 to 428). Tetanus is a pure toxemia; the bacilli or their spores may remain in the tissues without producing symptoms, if toxin is not formed. Not all strains of the tetanus bacilli produce toxin and it must be remembered that symbiosis of the spores of the bacilli with other organisms is necessary to produce toxin.

The toxin ascends the peripheral nerves to the spinal cord by way of the axis cylinders and the lymph channels of the epineurium and perineurium Robertson's experiments have further shown that the toxin reaches the spinal cord by way of the motor roots. Tetanus dolorosus may be caused experimentally by injecting toxin into the sensory nerve roots; the animals will die of exhaustion before motor phenomena appear. Experimental tetanus in the small laboratory animals is usually of the ascending type in which the symptoms appear first in the inoculated extremity. In the larger animals and in man the descending type is the rule; the characteristic spasm develops first in the muscles of the jaw and neck no matter where the site of injection. This is explained by the rapid absorption of the toxin into the general circulation. The toxin in the spinal cord stimulates the motor cells and makes the sensory system more susceptible to external stimuli. Tonic and clonic spasms of the muscles are then produced.

The treatment of tetanus may be divided into prophylactic and active. The most important factor in prophylactic treatment is thorough mechanical and chemial leansing of all wounds. Anti-tetanic serum should be given routinely to patients with contused and lacerated wounds which have been exposed to contamination. The results of the prophylaxis have been demonstrated during the recent war, as the mortality from tetanus in the A. E. F. was less than 2 per 100,000. The usual prophylactic dose of 1500 units may be given subcutaneously or, preferably, intramuscularly, as soon as possible after the injury; it should be repeated at intervals of eight to ten days until the wound is clean and granulating. The author also emphasizes the importance of giving serum before late operations on the injured areas. It is understood that active surgical care of the wound itself is not to be neglected.

The active treatment of tetanus has for its aim (1) removal of the source of the toxin, (2) neutralization of the toxin already formed, (3) depression of the function of the cord, and (4) careful nursing. The serum may be given subcutaneously, intravenously, or intraspinally. The subcutaneous method is the least satisfactory, because of the large amount of serum necessary to produce the desired effect. The injection should be given early and as nearly as possible in a single massive dose; moderate doses should be repeated until the symptoms begin to subside. Bromides and chloral have been found the most useful drugs to depress the spinal cord and control the convulsions.

The author reports 18 cases of tetanus with a mortality of 35.8 per cent following unsatisfactory treatment. Of 13 remaining patients to whom he was able to give early and efficient treatment, two died, or 15.3 per cent. J. I. MITCHELL.

SURGICAL ASPECT OF CHARCOT JOINT AND OTHER SYPHILITIC BONE AND JOINT LESIONS: F. J. Cotton (Ann. Surg., 1920, 72, 488-493). The author reports ten cases of syphilitic bone and joint lesions and believes much benefit may be derived from efficient fixation and orthopedic measures, combined with intensive anti-syphilitic treatment.

Six of the patients were tabetic with Charcot joints of the knee, wrist, ankle, thumb, and great toe, and showed definite improvement under treatment; two of these were classed as transitional in type, and two as syphilis of the bone.

The four remaining cases showed retrogression of symptoms and repair of soft tissues, but no regeneration of bone was detectable by the x-ray. Joint changes are arrested by treatment and bone repair instituted. Charcot joints have been considered incurable. Although they may not be completely curable, restoration to safe use is possible. No spirochaetae were found in the joint fluid, but in spite of this Cotton believes the lesions specific.

The treatment described by the author produces a prompt effect on the joint. The destruction is checked and bone is regenerated. The joint capsule shrinks and tightens. The excess of soft tissue and waste fluid is absorbed.

Two cases were operated on and the wound healed perfectly. From this it is thought that they may be made safe for ankylosing. J. I. MITCHELL.

TWELVE CASES OF THROMBOSIS OF THE CAVERNOUS SINUS: J. Julian Chisolm and S. Shelton (Archives of Surgery, Nov., 1920, P. 482). The rarity of the appearance of thrombosis of the cavernous sinus warrants a careful study of the

cases reported in the literature for a thorough understanding of the condition. Chisolm and Shelton record in full 12 cases, 8 of which were gleaned from the surgical records of 50,000 cases appearing at the Johns Hopkins Hospital during the past 20 years.

Etiology: The most common causes for thrombosis of the cavernous sinus are trauma, marasums and infection. Marasmic thrombosis arises from debilitating conditions either in the young or the old and is never primary, appearing secondary to a longitudinal or lateral sinus thrombosis.

In traumatic thrombosis the cavernous sinus becomes involved through fractures of the base of the skull, direct injury of the sinus, wounds through the orbit or during operations on the Gasserian ganglion.

Septic thrombosis, by far the most common form almost always arises secondary to a thrombosis of some of the efferent or afferent veins. It may arise primary to a basalar meningitis, abscess of the post-orbital space, suppurative sphenoiditis or cares of the petrous portion of the temporal bone.

The bacteria most often reported are staphylococcus albus, staphyloccoccus aureus, streptococcus pyogenes and the pneumococcus and diplococcus intracellularis.

Pathology: Marasmic thrombi are sterile; as a rule the same is true of traumatic and operative cases. Septic thrombi as elsewhere begin with phlebitis. At first the thrombus is red and fragile, adherent to its walls and nearly always fills the lumen. It may become purulent. The wall of the vessel is thickened and lusterless and often perforated. Usually there is involvement of other sinuses with both cavernous sinuses implicated. Often there is present a basalar meningitis of a purulent nature complicating the condition. Other complications are meningeal hemorrhages, a serious meningitis of Quinke, brain abscess, emboli of the lungs and other parts of the body, infarction of the lungs, liver, spleen, and kidneys. Usually there is more or less infection of the neighboring vessels and nerves, frequently necrosis of the sphenoid and the petrous portion of the temporal bone.

Symptoms: The history of a chill, severe headache, septic temperature and exophthalmos always beginning on one side but involving the other in from 24 to 48 hours immediately calls to mind thrombosis of the cavernous sinus as the most likely diagnosis. The temperature is usually septic in character. The primary infection may be slight e. g. a small furuncle of the face, infected hair follicle in the vestibule of the nose, etc. With the involvement of but one eye conditions that may similate thrombosis of the cavernous sinus are erysipelas, cellulitis of the orbit secondary to accessory sinus disease, tumors of the orbit, and arteriovenous fistula of the internal carotid artery, also tenonitis and thrombophlebitis of the ophthalmic veins.

Treatment: Many prefer to treat the symptoms as they arise, others adovate prompt and radical operations on the cavernous sinus. Statistics show that but 7% recover without operation. It should be added that with operation less than 7% recover. The various operations advocated for the relief of thrombosis of the cavernous sinus are the temporal route of Dwight and Hartley, the orbital route of Mosher, the ethmosphenoidal route of Langwoethy and the Luc operation through the antrum of the opposite side.

PAUL D. BEMSFORD.

GYNECOLOGY AND OBSTETRICS

SUPERVISORS:
ARCHIBALD L. McDONALD,
FIDELITY BLDG., DULUTH.
ALBERT G. SCHULZE,
LOWRY BLDG., ST. PAUL.

PUERPERAL SEPSIS AND ITS TREATMENT: R. S. Titus (Boston Med. and Surg. Jour., Sept. 23, 1920). This subject, which should be of interest to all readers, is so thoroughly and convincingly presented that it might be of value to present the author's conclusions which are as follows:

"Puerperal infection is bacterial infection. Organisma gain entrance, first by vaginal examination; second, by the presence of an abnormal vaginal flora at the time of labor; third, by extension from foci of infection elsewhere in the body. The three types of puerperal infection depending upon the organism are, first, hemolytic streptococcal infection; second, pyogeni infection; third, saprophyti infection. The treatment of each type depends upon the bacterial reaction and the knowledge of its pathology. By and large leave it alone. The curette is dangerous. The antiseptic douche unintelligent. The conservative treatment rational and harmless. Surgery plays its part only when the infection has produced localized pus, and then the oldtime surgical principle of the evacuation of pus obtains. Vaccines and serums have had their try and have been found wanting. The future holds its hope first in aseptic preparation of the patient, second, absolute cleanliness of the operator. In this way to lower the percentage of infection to its lowest possible point. Next, conservative treatment, to do no harm rather to go on the supposition that some good may come from interference and in the specific serum for each individual case based upon the success of the treatment of type four in pneumonia. Theoretically, vaginal smears and cultures of a pregnant woman taken soon before labor may point out those which contain harmful bacteria and in consequence, some method of making these harmful bacteria harmless may be evolved."

ALBERT G. SCHULZE.

A DEMONSTRATION OF CERTAIN TRANSITION STAGES FROM BENIGN TO MALIGNANT CONDITIONS IN THE OVARY, UTERUS AND VULVA: Thomas Watts Eden (Am. Jour. of Ob. and Gyn., Vol. 1, No. 1). 1. Such changes are gradual and it is

difficult to describe a definite precancerous stage. 2. In the ovary, cancer occurs usually in cysts and not in unaltered organs. Therefore, observance of the accepted rule of removal of all ovarian neoplasms is safe. 3. From the fundus of the uterus, he shows a typical histological picture of irregular prolifieration of the epithelium in gland tubules, which is regarded as precancerous, and indication for hysteretomy. 4. Erosions of the cervix may heal spontaneously or as a result of treatment by a replacement of the columnar epithelium and glands, by squamous cells. On the other hand, erosions accompanied by deep lacerations with round cell infiltration may become cancerous. This type of lesion should be freely excised, though hysterectomy is not indicated. In nulliparous women erosions remain more benign. 5. Cervical adenomatous polyps do not represent a precancerous state, but the rule of excision is a good one. 6. Chronic vulvitis is not necessarily precancerous, though leucoplacia may be so regarded, also vulvitis accompanying glycosuria.

The new "American Journal of Obstetrics and Gynecology" is of great promise to all physicians who are interested in these branches of medicine. The American Gynecological Society, The American Association of Obstetricians and Gynecologists, and the Obstetrical Societies of New York, Philadelphia, and Brooklyn, are represented in this publication. This fact together with the personel of the editorial board, guarantees an abundance of highgrade material. The reader may be assured that here will be found not only the best, but all of the substantial contributions in these subjects. Certainly, Volume 1. Number 1. is a worthy example and the list of accepted articles is a splendid indication for the future.

ARCHIBALD L. MCDONALD.

FURTHER OBSERVATIONS ON THE FUNCTION OF THE CORPUS LUTEM: E. H. Ochsner (Surg. Gyn. and OB., Vol. 31, No. 5). Dr. Ochsner considers some important relations of the corpus luteum to sterility, pregnancy abortion, and intra-abdominal hemorrhage; based on the experience of veterinary surgeons with cows, and on his own clinical records. 1. The presence of an unabsorbed false corpus luteum prevents "heat" in cows, and therefore the possibility of pregnancy. Uterine or periuterine infection delays such absorption and renders the animal sterile. This may be overcome by; (a) clearing up the local infection, or (b) removal of the unabsorbed corpus luteum. A number of clinical cases are cited which amplify the truth of this statement as regards the human. 2. Regardless of the date of the last menstruation, all women menstruate within 36 hours after surgial removal of an unabsorbed false corpus luteum. 3. The corpus luteum in the cow is not absorbed during suckling, hence "heat", and the possibility of pregnancy is not re-established. This period may be cut short by expression of the corpus luteum. This observation may explain the period of postpartum amenorrhoea and sterility in the human. 4. Injury to the true corpus luteum causes abortion in a large number of instances. This is more important than any other factor in laparotomy during pregnancy. 5. Rupture of an atypical corpus luteum may cause pelvic hemorrhage and uterine bleeding, simulating extra-uterine pregnancy.

ARCHIBALD L. MCDONALD.

PEDIATRICS

SUPERVISORS:
FREDERICK C. RODDA,
SYNDICATE BLDG., MINNEAPOLIS.
ROY N. ANDREWS,
MANKATO CLINIC, MANKATO.

NERVOUS AND UNSTABLE CHILDREN: E. Bosworth McCready (Arch. of Ped., Oct., 1919). The well poised, effective, emotionally stable adult human being is the exception rather than the rule in modern life. The neuroses and psychoses are not of sudden onset in but few instances do they develop in a previously sound individual. Some children are born nervous (hereditary causes); some acquire nervousness (disease, habits, etc.); others have nervousness thrust upon them (faulty training at home and at school, and by other social causes). To counteract and to remove these causes in their various phases, is, the duty and privilege of the physician.

The proper development of the nervous system is dependent upon proper metabolic functioning. Anything that interferes with this as, improper food, insufficient food, acute or chronic disease, fatigue, physical and emotional causes, lays the foundation for future trouble. In fact so closely associated and interrelated are the symptoms of deficient growth, energy and nervous insufficiency that the mutual and reciprocal action of each is apparent.

Treatment should begin as early in life as possible, employing every means to allow mature every opportunity for beneficient activity. Unfortunately the average private home, is conducted for the convenience of the adult members of the household, and the children may fit in as best they can, just so they keep out of the way of their elders. Thus the treatment of the nervous child must begin with a modification of the immediate environment. Again, no matter how solicitious and conscientious the parents may be, they lack the special training and experience which would enable them to deal effectively with a mal-adjusted child. The first consideration should be to see that the child obtains sufficient rest. Most children are over-stimulated by the conditions of modern life. Fatigue in children brings about a nervous irritability which leads to further effort, giving the impression that the child is unusually energetic. Equalling the importance of the prevention of over-fatigue is the regulation of the diet. The school child, in the habit of sleeping until the last minute, comes to the table with time only to bolt one or an-

other emasculated and widely exploited cereal whose only virtue is that it requires a large quantity of cream to be swallowed.

Fresh air, bathing, regulated exercise, all have an important place in the hygienic management of the nervous child. To the urban realist "pigs is pigs," but to the up-to-date farmer good pigs and other animals are balanced rations, exercise and fresh air. Our domestic animals are important and valuable, but are they more important and valuable than our children?

The Standard Dictionary states: "It (education) includes not only the narrow conception of instructions to which it was formerly limited, but embraces all forms of human experience, owing to the recognition of the fact that every stimulus with its corresponding reaction has a definite effect upon character."

ROY N. ANDREWS.

CYANOSIS IN THE NEW BORN: Frank Cohen, (Arch. of Ped., Nov., 1920) discusses cyanosis in the new born as an entity in itself and classifies around it the conditions under which it occurs. He describes cyanosis as "a condition of increased oxygen unsaturation in the peripheral capillaries." Oxygen unsaturation he defines as "the difference between the oxygen in the venous blood and the total amount of available oxygen in the blood."

Cyanosis occurs in the new born because of (1) the character of labor, (2) an abnormal developmental process, or (3) sepsis.

1. Asphyxia neonatorum is followed by cyanosis, as in cases of prolonged labor, cord about the neck, premature separation of the placenta, placenta previa, prolonged anesthesia, the asphyxia of the new born of twilight sleep, or that following pituitrin, and in intracranial or cerebral hemorrhage. In all but hemorrhage the cyanosis passes away with the proper treatment of the asphyxia.

2. Cyanosis is present in cases of abnormal development as pulmonary atelectasis, congenital heart defects, thymus hyperplasia, diaphragmatic hernia and other anomalies. In congenital heart defects cyanosis may be the only apparent sign, or if combined with the presence of a murmur will point very strongly to a cardiac defect. The extreme grades of cyanosis appear in pulmonary obstruction, in cases of pulmonary stenosis or atresia, and in the transposition of great vessels. With thymus enlargement, the cyanosis may be intermittent, or continuous, and may be accompanied by inspiratory stridor, or convulsions. In cases of diaphragmatic hernia there is an extreme grade of cyanosis and dyspnea with signs of cardiac displacement, absence of murmurs, and absence of pulmonary resonance on one side.

3. Cyanosis may be a prominent symptom in infectious cases such as peritonitis, pneumonia, arthritis, osteomyelitis, septic conditions following cord infections and the like. N. O. PEARCE.

FOR SALE—Country practice, village of 400, good farming community. Worth $4,000 annually without surgery—collections A-1. Am retiring on account of health. Will vacate immediately in favor of the purchaser of my office equipment at a very reasonable figure. Address AMR c-o Minnesota Medicine.

WANTED an assistant or an associate by Eye, Ear, Nose, Throat man in Twin Cities. Address, XYZ care MINNESOTA MEDICINE.

PHYSICIANS' MICROSCOPE
FOR SALE
In first-class condition. Apply
FROST'S DRUG STORE, Selby and Western Avenues
ST. PAUL, MINN.

Fine opening for Hospital in fair sized Minnesota town in center of rich farming section about sixty miles from Twin Cities. Excellent building available, all modern conveniences, large grounds. No competition. For particulars address A. B. C., care MINNESOTA MEDICINE, Saint Paul.

Three-Year Fellowships in Internal Medicine
With Living Stipends Are Available, Service Beginning April 1st, July 1st and Thereafter in
THE MAYO FOUNDATION FOR MEDICAL EDUCATION AND RESEARCH
ForFurther Information, Address the Director ROCHESTER, MINNESOTA